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VOLUME 6 NUMBER 3

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CONTENTS OF PREVIOUS NUMBERS

JANUARY, 1923. NUMBER 1—PART I

The Bile Factor in Pancreatitis. Frank C. Mann, M.D., and Alfred Giordano, M.D., Rochester, Minn.

End-Results of Five Hundred Cases of Chronic Appendicitis: A Statistical Study. John B. Deaver, M.D., and I. S. Ravdin, M.D., Philadelphia.

Studies in Experimental Traumatic Shock: VI. The Action of Ether on the Circulation in Traumatic Shock. McKeen Cattell, Ph.D., Boston.

The Roentgenologic Aspect of Chronic Appendicitis. Henry K. Pancoast, M.D., Philadelphia.

The Relation of the Heart, Pericardium and the Heart Valves to the Anterior Chest Wall. Leon T. LeWald, M.D., New York.

The Value of Pericardiotomy in Diagnosis and Treatment. John B. Roberts, M.D., Philadelphia.

Chronic Noninflammatory Lesions of the Knee Joint. Melvin S. Henderson, M.D., Rochester, Minn.

Gastrojejunal Ulcer: An Experimental Study. Albert H. Montgomery, M.D., Chicago.

Mesenteric Vascular Occlusion. Leo Brady, M.D., Baltimore.

Bilateral Chronic Open Pneumothorax Cured by Negative Tension: Report of Case. E. M. Eberts, M.D., Montreal, Canada.

Outline of Treatment of Fractures: Syllabus Adopted at the Boston Conference, April, 1922.

Nineteenth Report of Progress in Orthopedic Surgery. Robert B. Osgood, M.D.; Robert Soutter, M.D.; C. Hermann Bucholz, M.D.; H. C. Low, M.D.; Murray S. Danforth, M.D.; Lloyd T. Brown, M.D.; Philip D. Wilson, M.D., and Armin Klein, M.D., Boston.

JANUARY, 1923. NUMBER 1—PART II

Transactions of American Association for Thoracic Surgery

Officers for 1921-1922.

Officers for 1922-1923.

The Present and Future in Thoracic Surgery. Samuel Robinson, M.D., Santa Barbara, Calif.

Surgical Treatment of the Esophagus. Hermann Fischer, M.D., New York.

Posterior Mediastinotomy. Howard Lilienthal, M.D., New York.

Teratoma of the Right Chest Cavity: Report of Case. Wyman Whittemore, M.D., Boston.

A Radium Needle for the Esophagoscope. Sidney Yankauer, M.D., New York.

Surgery of the Mediastinum, Including the Heart and Esophagus: Abstract of Discussion.

The Differential Diagnosis Between Tuberculosis and Lung Abscess: A Clinical and Roentgenologic Study of Twelve Cases Seen at St. Luke's Hospital. Leon T. LeWald, M.D., and Nathan W. Green, M.D., New York.

Lung Abscess. A. L. Lockwood, M.D., Toronto, Ont.

The Surgical Treatment of Bronchiectasis with a Report of Three Cases of Removal of a Lobe of the Lung. Evarts A. Graham, M.D., St. Louis.

Lung Abscess. G. J. Heuer, M.D., Cincinnati, and P. M. MacCready, M.D., Baltimore.

The Interrelationship and End-Results of Chronic Suppurative Diseases of the Lung. Willis S. Lemon, M.D., Rochester, Minn.

Fibrosis of the Lung Following Ligation of the Pulmonary Artery Combined with Phrenicotomy, and Following Partial Occlusion of the Pulmonary Veins. Karl Schlaepfer, M.D., Baltimore.

Observations on Lung Suppuration and Its Treatment: A Brief Review of Personal Experiences and Clinical Impressions Obtained Within the Last Twelve Years. Willy Meyer, M.D., New York.

Proceedings.

List of Members of the American Association for Thoracic Surgery.

MARCH, 1923. NUMBER 2

Benign and Malignant Growths of the Nasopharynx and Their Treatment with Radium. S. J. Crowe, M.D., and John W. Baylor, M.D., Baltimore.

The Etiology of Urinary Lithiasis: An Experimental Study. Linwood D. Keyser, M.D., Rochester, Minn.

The Nature of Cranial Hyperostosis Overlying Endothelioma of the Meninges. D. B. Phemister, M.D., Chicago.

Osteoma of the Skull: Report of Two Cases, One Being Associated with a Large Intracranial Endothelioma. Carl W. Rand, M.D., Los Angeles.

Studies in Exhaustion: IV. Physical Trauma. G. W. Crile, M.D., Cleveland.

Alterations in the Currents and Absorption of Cerebrospinal Fluid Following Salt Administration. Frederic E. B. Foley, M.D., St. Paul.

The Relation Between Oblique Inguinal Hernia and Workmen's Compensation Laws. J. M. Wainwright, M.D., Scranton, Pa.

Pathogenic Ptosis of the Right Colon. E. P. Quain, M.D., Bismarck, N. D.

Carcinoma of the Appendix. Arnold S. Jackson, M.D., Rochester, Minn.

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ARCHIVES OF SURGERY

VOL. 6 MAY, 1923 No. 3

CYSTIC DISEASE OF THE BONES

A STUDY OF FIFTEEN CASES

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I. CLINICAL REPORT BY DR. ASHHURST

Diseases of any tissue in the body may be classified conveniently into three general categories: infections, dystrophies and tumors. But while under each category there are perfectly typical cases, there exist other cases of disease concerning the classification of which there may be uncertainty or disagreement, not to say dispute. The existence of such cases is particularly noticeable in connection with bone diseases, and it is to illustrate this difficulty that the present report of a series of cases of cystic disease of the bones is presented. The only common feature, in some instances, is that in the interior of the bone there existed disease which caused rarefaction, absorption or destruction of bony tissue, with, in many cases, expansion and thinning of the overlying cortex. The causes are various: pyogenic bacterial infection, tuberculosis and syphilis, as well as unknown causes including a number of cases of true tumor formation.

1. *Infections.*—Let us take first, for example, a case of ordinary hematogenous staphylococcic infection of the bones. In its acute form this infection produces typical acute osteomyelitis; in its chronic form (usually a recurrence or a sequel of an osteomyelitis formerly acute) it produces a scarcely less typical form of chronic osteomyelitis, attended rarely by sequestration, but often by great thickening and hardening of the bone, with a destruction of bone which is slight or almost absent: such cases sometimes take the form of a Brodie's abscess (1824), a Markoe's abscess (1872), or merely of bone sclerosis without demonstrable pus or central cavity (Garré, 1893).[1] In the subacute

1. An example of this condition, which is not so rare as has been thought, was reported by the author previously (Med. & Surg. Rep. Episc. Hosp. **4**:110, 1916, Case 5).

type, however, a definite cystic area, not resembling a Brodie's abscess, may develop in the bone affected (Case 1).

But, in addition to frank pyogenic infections, there is also recognized a class of diseases grouped together under the title of infectious granulomas (tuberculosis, syphilis, actinomycosis, etc.), in each of which a more or less characteristic granulation tissue is produced. When occurring in bone, these diseases, especially tuberculosis, may take on a cystic character, and, except by pathologic examination of the tissue removed from the cyst, a correct diagnosis between these and other forms of bone cysts cannot always be made. In Case 2, for example, the disease was in all probability tuberculous, but owing

Fig. 1.—Acute syphilitic periostitis; subperiosteal gumma.

to the absence of a pathologic report this is not certain. In Case 3, however, both pathologic examination and the clinical course of the disease demonstrated the tuberculous nature of the infection. It is interesting to note that a case, apparently the exact counterpart of this so far as the roentgen-ray appearances are concerned, has been recorded by Barrie [2] as a case of chronic fibrocystic osteomyelitis.

The only cystic lesions I have recognized in bone as being due to syphilis are of two kinds: those occurring under the periosteum in the acute stages of syphilitic periostitis (Fig. 1), and those seen in the short bones and in the ends of the long bones in certain cases of the hereditary form of the disease. A notable example of the latter was illustrated in a patient under my care some years ago, in whom the

2. Barrie: Surg. Gynec. & Obst. **19**:42, 1914 (Fig. 2).

expanded cystic ends of the long bones and the extremely cystic carpal and tarsal bones represented an abnormal degree of eccentric atrophy (Fig. 2). The history of this patient was narrated in detail by Dr. John P. Jones [3] in 1915. The patient eventually became able to walk without braces, and when last seen (about seven years later) was in good general physical condition.

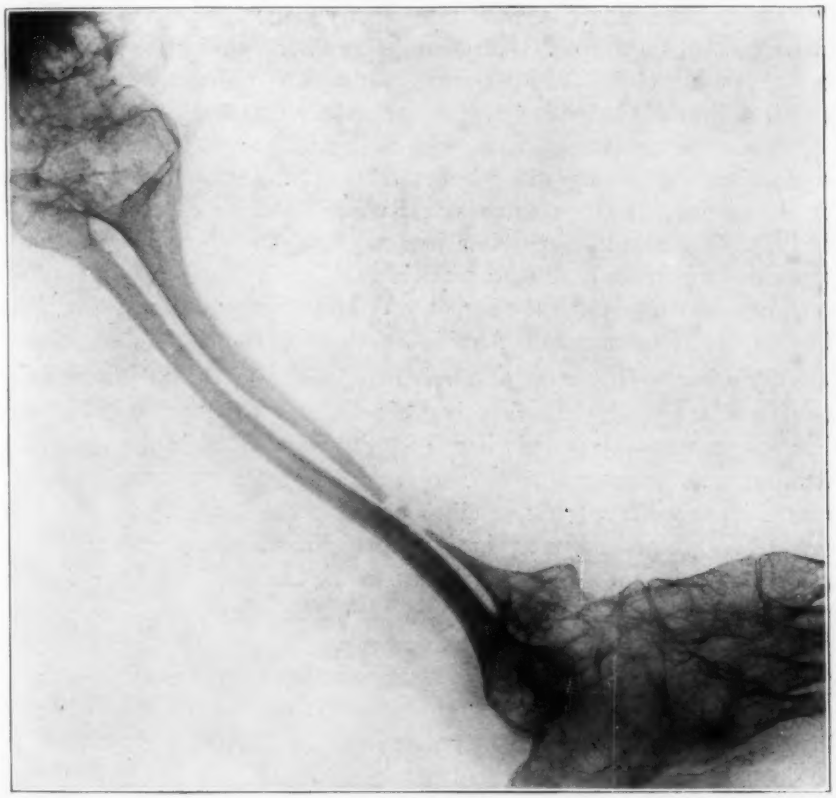

Fig. 2.—Hereditary syphilis, with cystic changes in the ends of the long bones and in the tarsus.

2. *Dystrophies.*—These are lesions which may be due to very remote or attenuated infections, or to alterations or congenital abnormalities of metabolism, but whose true cause often remains undetermined. All that can be safely said is that they are due to disturbances of nutrition. In the autumn of 1914 there came under my care, at the Episcopal Hospital, within a few weeks of each other, two young English patients with recurrent deformity of one lower extremity (one knock-knee, the other bow-leg) developing some years after the opera-

3. Jones, J. P.: Med. & Surg. Rep. Episc. Hosp. **3**:68, 1915.

tive correction of similar deformities. In both, the affected bone was markedly atrophied (eccentric atrophy) and cystic (Cases 4 and 5). Except for possible malnutrition in childhood following the operation, or as a sequel of the operative injury, there appears to have been no adequate explanation of the cystic disease. The elder of the two, a man 25 years of age, was a vegetarian. Possibly this accounted for the dystrophy.

Much more usual than the cases just recorded, but possibly of the same general type, are those known in the literature by the terms bone cysts, cystic osteitis, osteitis fibrosa cystica, etc. Their pathogenesis is still in dispute. Case 6 is a typical example.

Again, of the same general type, but quite rare, are instances of the same disease affecting many bones at the same time—multiple fibrocystic osteitis, known also as von Recklinghausen's disease (Cases 7 and 8). The relations of this affection to Paget's disease of the bones (osteitis deformans), to osteomalacia and kindred affections in adults remains obscure, though the recent very interesting studies of Dr. E. P. Corson White [4] throw considerable light on the subject.

3. *Tumors.*—The term tumor is here used in a clinical sense, inasmuch as the pathologic and pathogenic sense of the term is in dispute. From an academic point of view, I admit that any cell in the body may form a tumor, and that this tumor may be of the embryonal (malignant), intermediary (questionable) or adult (benign) type. The two extremes (benign and malignant) are not difficult to recognize; but, in accordance with the dictum "Natura non facit saltus," there are a great many tumors of intermediary type, which, as Ewing says, "are neither benign nor malignant." And then there are lesions which are neither granulation tissue (inflammatory reaction) nor yet true tumors. Thus, at the end of the tumor classification which is nearest the class of cases just discussed, especially to that illustrated in Case 6, I place Case 9, an instance of "cystic osteitis with giant cells," as our pathologist named it. Next to it (Case 10), I place a case of "giant cell sarcoma" (myeloma) [5] which returned after primary evacuation, then, under the

4. White, E. P. C.: Osteitis Deformans in Monkeys, Arch. Int. Med. **30**: 790 (Dec.) 1922.

5. The term myeloma I prefer to that of giant cell sarcoma or giant cell tumor. The term myeloplaxoma, originally chosen by Eugene Nélaton (1860) to designate this benign tumor, was abbreviated into myeloma by Malherbe in his book on sarcoma (1904); and the latter term is in common use in Great Britain and Canada, as well as in France. It is intended to describe a benign tumor of bone marrow. A malignant tumor of bone marrow is a sarcoma. A tumor of intermediary type may well be called a myeloid sarcoma (Case 13). A myeloma should, of course, be distinguished from the general disease called myelomatosis or multiple myeloma, with which the benign tumor myeloma has no connection.

influence of Coley's toxins, swelled up larger still, ruptured, discharged its contents and gradually became condensed until only a small bony tumor remained. Following this comes a myeloma (Case 11), which undergoing favorable evolution cured itself without any operative treatment; while Case 12 represents, on the contrary, a patient with a myeloma which recurred in semisarcomatous form after evacuation, but who is now well nearly nine years after radical excision of the recurrent disease. Here we appear to have progressed from the least neoplastic type of growth (Case 9) to one which becomes semimalignant (Case 12).

A closely similar condition, clinically, was seen in Case 13; but the mere fact that it occurred in a much older patient made us suspect that clinically it might be a malignant process, and our pathologist at first classed it as a spindle cell sarcoma, but later revised his diagnosis to "giant cell sarcoma of bone marrow with areas showing early spindle cell sarcoma." [6]

Finally, to round out the series of cystic tumors, I include an instance of chondroma undergoing cystic change (Case 14) and a case of cystic disease of the tarsal scaphoid (Case 15) grossly resembling a myeloma, but proved by microscopic study to be an angioma.

II. INTERPRETATION OF ROENTGENOGRAMS OF CYSTIC DISEASE OF THE BONES BY DR. BROMER

When a roentgenologist is called upon to make a diagnosis of a cystic condition in bone on the roentgenogram negative, the mere state-

6. In regard to the presence of spindle cells and round cells in bone cysts, Bloodgood (J. Radiol. **1**:168 [March] 1920) remarks that in almost all of his cases of bone cysts with osteitis fibrosa he has found cellular areas with spindle cells or round cells. The spindle cells are apparently of the connective tissue type which ultimately form fibroblasts and fibrous tissue, and the round cells are either osteoblasts which have not yet formed bone or cells which arise from proliferation of the endothelial cells of blood vessels. In a number of his reported cases in which tissue was sent to the laboratory for diagnosis, a diagnosis of sarcoma was made from the frozen section, while further study and the patient's clinical course showed the lesion to be benign. Similarly, our pathologist, Dr. C. Y. White, has frequently called our attention to perfectly typical areas of spindle cells from which, alone, any pathologist would make a diagnosis of sarcoma; yet study of other areas in the same section, and of sections cut from other blocks, rendered the diagnosis of sarcoma very doubtful or entirely precluded the idea of malignancy. Dr. White employs the convenient terms "giant cells" and "gigantic cell," to describe, by the former, the ordinary epulis type of multinucleated cell named myeloplax by Eugene Nélaton (1860), the first writer to describe systematically as a benign non-sarcomatous tumor the "giant cell sarcoma"; while by the term "gigantic cell," Dr. White designates the multinucleated "true tumor giant cell," which by many is considered indicative of malignancy, being found in carcinoma as well as in sarcoma.

ment that rarefied areas, a cystic process, exists is neither satisfactory to himself nor to the surgeon. If the diagnosis of the causative factor is difficult for the surgeon to make clinically, it is equally difficult for the roentgenologist and the same might be said for the pathologist. It has been my experience, after a diagnosis of sarcoma had been made from the roentgenograms, to have the pathologist return a report on the excised specimen of chronic inflammatory tissue. Six months afterward, he returned a report of spindle cell sarcoma, when a second excised specimen had been sent to him. Further cutting of the first specimen revealed the same cells in it as were later found in the second.

The purpose of the presentation of these roentgenograms is to emphasize the impossibility, at times, of an exact roentgen-ray diagnosis. If nothing original or new is advanced, it seems well worth the effort to reproduce roentgenograms of varied lesions showing striking similarity, for by close study of them something of importance is gained.

Dr. Ashhurst has generally classified the foregoing lesions in three categories: infections, dystrophies and tumors. Lovett [7] has pointed out the impossibility, in the case of infections, of differentiation of syphilis, chronic osteomyelitis and tuberculosis, by means of the roentgen ray alone. Bloodgood [8] has shown the impossibility of such differentiation in the case of tumors, especially cysts, giant cell tumors and central sarcomas. Ewing [9] has pointed out the difficulties of the pathologist.

The more one reads the existing literature, the more disheartened he is as a result of the lack of assistance. Numerous classifications abound. Baetjer [10] has attempted to aid by his laws of possibilities, age, etc.; but their practical application, when one is confronted by the actual lesion, is not easy. It is no bold assertion to say that every roentgenologist feels his limitations, in this respect.

It is our plan to set forth, for each roentgenogram, explanatory notes, stating the points of similarity, difficulties in diagnosis and any other characteristics which serve to strengthen or confirm our opinion of the exact underlying pathologic process in each lesion.

REPORT OF CASES

CASE 1.—*Osteomyelitis of shaft of humerus resembling cystic disease. History.*—C. R. C., a red haired youth, aged 19, came under my care in

7. Lovett, R. W., and Wolbach, S. B.: Roentgenographic Appearance, Diagnosis, and Pathology of Some Obscure Cases of Bone Lesions, Surg., Gynec. & Obst. **31**:111 (Aug.) 1920.

8. Bloodgood, J. C.: The Diagnosis and Treatment of Benign and Malignant Tumors of Bone, J. Radiol. **1**:147 (March) 1920.

9. Ewing, James: A Review and Classification of Bone Sarcomas, Arch. Surg. **4**:485 (May) 1922.

10. Baetjer, F. H., and Waters, C. A.: Injuries and Diseases of the Bones and Joints, New York, Paul B. Hoeber, 1921.

the Episcopal Hospital, Sept. 14, 1921, complaining of pain and swelling of the right arm. He had been in the hospital on numerous occasions previously, the first time in the summer of 1914. At that time, when 12 years of age, he was admitted to the medical service with the diagnosis of typhoid fever; but staphylococci were grown in cultures made from his blood, and eventually he was transferred to my care with pains in the right sacro-iliac region and hip. After rest in bed with weight extension for about four months, he was discharged apparently well, no local lesions having developed. About a year later, in January, 1916, an abscess developed and later discharged over the right sacro-iliac joint. Because of this condition he was readmitted to the hospital. Although there seemed to be free drainage, he continued to have a hectic rise of temperature for a week after admission, so operation was undertaken. Roentgenographic examination after bismuth injection of the sinuses revealed that one apparently extended to the right acetabulum; but the clinical diagnosis was necrosis of the sacrum. At operation (Jan. 21, 1916), I traced one sinus to the region of the great trochanter; but it led to no bone lesion. Another sinus was followed to the lateral border of the sacrum from which a few small cortical sequestrums were removed. The wound was swabbed with pure phenol and packed with gauze. Convalescence was uneventful, and he was discharged with the wound healed, two or three months later. More than a year later (in 1917) while at home, two sinuses developed in the right thigh near the hip and discharged a little pus for more than a year. He was again in the hospital for three months, under Dr. Gill's care (during my absence in France); but no operation was performed. The sinuses did not heal finally until 1919, and in September, 1919, he was again in the hospital and was treated with weight extension for three months because of pain. He went home with the sinuses still healed, and they have remained healed ever since. He has now had no symptoms referable to the hip for two years. During the year 1919, however, a lump on the anterior surface of the right arm above the elbow formed and again disappeared several times. It was finally lanced, and a spicule of bone was removed. The sinus healed in about a month, and his arm had given him no further trouble until recently.

In June, 1921, three months before the present admission to the hospital, he was hit on the right arm by a baseball, and some time later fell from a motor cycle and may have injured his arm. Two months before, he had some dull pain in the arm and was treated by his family physician for neuritis. The pain commenced again a week previously, and the arm began to swell. Then the pain diminished.

Examination.—On admission, Sept. 14, 1921, there were noted the scars of healed sinuses about the right hip, thigh and sacro-iliac region. The movements of the hip were normal and painless. The right arm was swollen, red and hot. It was not very painful even on deep pressure. There was palpable bony thickening in the middle of the shaft. About 2 cm. below this area, on the anterior surface of the arm, was the scar of a healed sinus. The roentgen-ray report by Dr. R. S. Bromer stated, "There is a fusiform swelling of the shaft with much periosteal thickening which is laid down longitudinally. There is widening of the medullary canal and a shadow suggestive of a sequestrum lying within it. I hardly think this is due to a new growth but to an osteomyelitis" (Fig. 3).

Operation.—Sept. 16, 1921, an incision, 20 cm. long, was made between the biceps and triceps, exposing the thickened roughened shaft with one cloaca

on its anterior surface affording admission to a cavity about 5 cm. in length in the medulla. No sequestrum was found. The involucrum was removed on the lateral surface of the humerus for a distance of 15 cm., both sides of the gutter being cut down until only half the thickness of the bone remained. The wound was wiped with picric acid (2 per cent. alcoholic solution) and closed

Fig. 3 (Case 1).—Staphylococcic osteomyelitis of humerus, subacute; abscess with cystic appearance.

with deep sutures of silkworm gut, with an iodoform gauze wick emerging at the lower angle, and the arm was bound to his side and a shoulder cap applied.

Course.—Oct. 1, 1921, the wound was nearly healed. There was a slight purulent discharge from the lower angle.

September, 1922, the wound broke open once, three months after operation, but healed in a week and has remained healed since.

March, 1923, there was normal function and no disability whatever.

CASE 2.—*Bone cyst of the tibia. History.*—C. G., a brunette Italian girl, aged 5 years, had always been well except for measles and whooping cough in infancy. For about two years before coming under observation, there had been some swelling around the left ankle: no history of injury was obtainable. Lately there had been much pain in walking.

Examination.—On admission to the Episcopal Hospital, Aug. 25, 1913, the soft parts over the lower end of the left tibia were swollen and tender. There was no redness and no heat. The tissues around both malleoli were puffed up,

Figure 4

Figure 5

Fig. 4 (Case 2.)—Cystic osteitis, probably tuberculous, of lower end of tibia.

Fig. 5 (Case 2).—Cystic osteitis, probably tuberculous, of lower end of tibia.

and there was marked tenderness on movement of the foot, especially on forced dorsal flexion. A roentgenogram (Figs. 4 and 5) revealed a bone cyst of the lower end of the tibia, the ankle-joint appearing normal.

Operation.—Aug. 27, 1913, under Esmarch anemia, a curved incision was made over the seat of disease, and a flap of skin and subcutaneous tissue turned aside. The periosteum was slightly thickened, and the cortex slightly hyperemic. The cortex was about 2 mm. thick, and when this was removed a yellowish white cyst wall bulged into the opening. When this was cut through with scissors, some semifluid matter resembling tuberculous pus, oozed out, and then considerable cheesy matter was evacuated, but no free fluid. There was a good deal of carious spongy red bone surrounding the cavity, and

it was thought to be a tuberculous abscess of bone (Brodie's abscess). On the posterior wall of the tibia, the cyst had perforated the cortex over an area about 3 mm. in diameter. The cyst cavity was the size of a pigeon's egg and extended down to the epiphyseal cartilage. The cavity was scraped clean and filled with iodoform bone wax (Mosetig-Moorhof); no drainage was established. The leg was dressed in plaster of Paris.

Course.—September 3, the gypsum dressing was removed because of pain in the leg. A little of the wax was discharging between two sutures. A new gypsum case was applied, with a window over the wound.

October 3, no discharge had come from the wound since the last note. The wound was now healed.

October 10, the patient was walking in a gypsum case.

October 15, the case was removed. Swelling over the ankle was less. A new case was applied without any window over the wound.

November 5, the case was removed. There was no swelling, and good motion in the ankle. A brace was ordered.

November 28, the patient was sent home walking well in the brace. A roentgenogram made before the patient went home revealed considerable concentric growth of bone around the iodoform wax plug, which had been slightly absorbed.

Feb. 12, 1914 (six months after operation), the leg looked practically normal. The ankle could not be dorsiflexed beyond 90 degrees. There were no symptoms. The patient walked well, and still used the brace. (It has been impossible to trace the patient.)

Pathologic Report (Dr. C. Y. White).—Culture and smear from the cyst were negative. (Unfortunately, the cyst wall and contents were mislaid in the operating room and did not reach the laboratory in fit condition for histologic study or for inoculation into guinea-pigs.)

Case 3.—*Tuberculous cyst of ulna. History.*—J. C., a brunette boy, when about 6 years old, fell and hurt his left elbow. He seemed to have suffered no particular disability as a result; but two years later (July, 1914) he applied to the orthopedic dispensary of the Episcopal Hospital because of limitation of motion.

Examination.—There was decided limitation of rotation in the forearm, and the elbow could be flexed only to 50 degrees and extended to 130 degrees. There was no pain, no heat and only slight thickening of the elbow region. A roentgenogram revealed a cystic condition of the upper end of the ulna, apparently extending to the joint surface (Fig. 6). It was thought to be a "benign bone cyst," and, after keeping the child under observation for four months and finding no change in the local condition, operation was advised and agreed to by the parents.

Operation.—Nov. 4, 1914, under Esmarch anemia an incision was made over the subcutaneous surface of the ulna below the olecranon. The periosteum was incised and reflected. The cortex appeared unduly inflamed, with a large number of bleeding points, and was soft and easily removed by a gouge, without the use of a hammer. The cortex was 0.5 cm. thick, and when it was removed a smooth fibrous layer was exposed, which constituted the cyst wall. Enough of the overlying cortex was removed to expose the cyst wall fairly well over an area approximately 2 by 4.5 cm. An attempt

was then made to enucleate the cyst without rupture; but this proved impossible since, at the coronoid process of the ulna and over the entire articular surface, the cyst wall was densely adherent to the bone, dipping down into irregular notches and depressions. Those portions of the cyst wall which were not removed with the cyst contents were scraped off the adjacent bone

Fig. 6 (Case 3).—Cystic osteitis, tuberculous, of upper end of ulna.

with the sharp spoon. The contents of the cyst resembled granulation tissue. No fluid was present. The cavity in the ulna was filled with iodoform bone wax, and the periosteum and then the skin were tightly sutured. The Esmarch bandage was not removed until after the dressings had been applied.

Pathologic Examination (Dr. C. Y. White).—This revealed tuberculous osteomyelitis. Injection of the tissue into a guinea-pig was positive for tuberculosis.

This report is of interest, especially as the roentgenogram did not suggest a tuberculous process. The roentgenogram is practically identical with that of the second case reported as chronic fibrocystic osteomyelitis by Barrie.[2]

Course.—November 23, there had been a little discharge of wax since operation.

December 7, the wound was firmly healed.

December 18, a plaster-of-Paris case was applied, the laboratory report of the tuberculous nature of the lesions having been received.

March, 1915, the gypsum case was removed and the arm carried in a sling.

May: As heat in the elbow persisted, an internal angular splint was applied, in addition to the sling.

June: There was no heat or swelling. The splint was removed.

July: Use of the sling was discontinued.

October: Motion of from 50 to 120 degrees was possible. There was never any pain.

August, 1916: Motion of from 70 to 140 degrees was possible. There were no symptoms.

November: Motion of from 45 to 115 degrees was possible. The patient played ball and had no disability. It was believed that if motion did not improve when epiphyseal growth was complete, excision of the elbow might be advisable. A roentgenogram revealed no vestige of wax remaining. The upper end of the ulna was rarefied, but much of the cortex had reformed, apparently of normal density.[11]

CASE 4.—*Fibrocystic osteitis of femur developing after operation for knock-knee. History.*—M. E., a brunette, aged 16 years, was admitted to the Episcopal Hospital in October, 1914, complaining of disability from recurrence of a knock-knee deformity in the left lower extremity. When 3 years of age she had been operated on in England for knock-knee, being told at that time that she had only cartilage, no bone, in the knee. The deformity gradually recurred.

Examination.—There was lateral movement in the knee joint through an angle of 50 degrees (from 180 to 130 degrees), and in flexion the patella luxated externally, entirely disabling the patient. The left lower extremity was 2 cm. shorter than the right. There were scars over the inner surface of the upper end of the tibia, and above the internal condyle of the femur, which she said were those of the operation, thirteen years previously. A roentgenogram showed a cystic area in the lower end of the femur.

Operation.—Oct. 24, 1914, an incision about 10 cm. long was made from the patella upward between the rectus and vastus internus muscles. The latter was cut through, exposing the femoral shaft. The quadriceps bursa was then raised from the femur, and a wedge of bone was cut from the median surface of the femur just above the internal condyle. The cortex was very thin, fracturing almost like an eggshell, and the marrow was almost fluid. Good correction of the deformity was secured, after removal of the wedge, by breaking the thin cortex on the lateral margin of the femur by adduction of the leg on the thigh. A plaster-of-Paris dressing was applied.

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11. During the author's absence in France (1917-1919), Dr. Gill performed an excision of the elbow for recurrence of tuberculosis.

Pathologic Report (Dr. C. Y. White).—No organisms were visible in a smear made from the fluid in the medullary cavity, and a culture showed no growth. A guinea-pig inoculation was negative for tuberculosis. Histologically, the sections from the marrow showed only chronic granulation tissue, becoming fibrous.

Course.—The gypsum case was removed two months after operation, but a brace was worn for a year, supporting the knee against undue strain. Five years and three months after operation, the patient was reported to be in good health, with no recurrence of the deformity and normal use of the knee.

CASE 5.—*Fibrocystic osteitis of tibia developing after operation for bow-leg. History.*—A. M., a brunette man, aged 25, was admitted to the Episcopal Hospital Oct. 24, 1914, complaining of instability in the left knee, which was subject to recurrent sprains. He had been operated on in England ten years, or more, previously for bow-legs, and for the last five or six years this deformity had been returning, especially in the left lower extremity where the bowing was quite marked, owing to outward curvature of the tibia.

Examination.—Roentgenograms showed changes in the head of the tibia suggesting cyst formation. The knee was bowed outward at an angle of 160 degrees; there was moderate (from 5 to 10 degrees) lateral motion in the joint, and the limb was 2.5 cm. shorter than its fellow.

Operation.—Nov. 6, 1914, a skin flap was turned up over the subcutaneous surface of the upper end of the left tibia, and the periosteum was retracted. Transverse osteotomy was performed about 6 cm. below the level of the knee joint, dividing the entire median surface of the tibia. The medullary cavity seemed to extend unduly far toward the bone end; it resembled currant jelly in appearance, but was more friable. A piece of bone, 2.5 by 1.25 by 1 cm., was cut from the cortex of the tibia shortly below the site of the osteotomy. This was inserted in the gap made by correcting the bow-leg deformity. The bone wedge effectually prevented its recurrence. A plaster-of-Paris dressing was applied.

Pathologic Report (Dr. C. Y. White).—Tissue removed from the marrow cavity, on guinea-pig inoculation, was negative for tuberculosis; but after sufficient tissue had been used for this purpose the specimen was not satisfactory for histologic examination.

Course.—Dec. 31, 1914, a brace was applied after the gypsum case had been removed. The knee was straight. Jan. 18, 1915, the patient went home wearing a brace, but walking without crutches. The patient has not been traced.

CASE 6.—*Bone cyst involving the upper end of the humerus. History.*[12]—J. R., a brunette boy, aged 12 years, fell and injured the right shoulder in the summer of 1911. He was treated for a sprain. He fell again in November, 1911, and broke the humerus. About ten days after this injury he was admitted to Dr. Frazier's service in the Episcopal Hospital, and came under my care. There evidently was an unreduced fracture of the upper end of the humerus. A roentgenogram confirmed the diagnosis; nothing else abnormal was noted at the time. Excellent reduction, as shown by another roentgenogram, was secured under a general anesthetic, and recovery of perfect function was uneventful.

About three months later (March, 1912), another roentgenogram was made to see whether there had been any permanent injury to the epiphyseal cartilage. There were now indubitable evidences of a change in the upper portion of the medulla of the humerus. The whole upper end of the diaphysis was thickened;

12. Compare Med. & Surg. Rept. Episc. Hosp. **1**:290, 1913.

two rarefied areas in the medulla were surrounded by denser bone, and there were indications of bony trabeculae crossing the rarefied areas. The functions of the joint were perfect; there was absolutely no deformity, and there were no subjective symptoms of any kind. For these reasons no treatment was instituted; but the boy was kept under observation. Reexamination of the original roentgenograms revealed thickening of the upper end of the humerus, probably indicating the presence of disease at that time (November, 1911).

In August, 1912, nine months after the injury, another roentgenogram was made. This showed the cystic change more developed, with thinning of the cortex. There were no subjective symptoms whatever, and all of the functions

Fig. 7 (Case 6).—Bone cyst, upper end of humerus, with pathologic fracture. (Courtesy of Lea and Febiger; from Ashhurst's "Surgery.")

of the shoulder joint, including external rotation and abduction, were fully preserved. On palpation, however, a distinct though slight thickening of the humerus could be detected, when compared with the other arm.

The boy fell again, Jan. 6, 1913, and injured the right shoulder. He came at once to the Episcopal Hospital and was seen by me. A roentgenogram (Fig. 7) showed a partial fracture of the cyst wall below the greater tuberosity. The parents then consented to operation.

Operation.—Jan. 10, 1913, the patient was etherized, and an Esmarch bandage was applied above Wyeth's pins, as for shoulder joint amputation.

An incision was made from the coracoid process down to the insertion of the deltoid muscle along the interspace between the deltoid and pectoralis major. The cephalic vein was left attached to the deltoid. Sterile gauze was then sutured to the skin margins of the wound. With another scalpel the intermuscular incision was deepened, and the periosteum was incised just to the outer side of the long tendon of the biceps. The periosteum was thickened, but no distinct new subperiosteal bone was present. The fracture on the outer side

Fig. 8 (Case 6).—Humerus, ten years after operation.

of the surgical neck was thus exposed; one shell of the cortex, as shown in the roentgenogram, stood out from the shaft. From this there was a faint line of fracture running transversely inward. No rotation of the lower fragment on the upper was possible.

The cortex, which was about 2 to 3 mm. thick, was then cut away with a gouge, exposing the medullary cavity which was filled with thin blood-stained fluid. This was sucked out with a syringe and sent to the laboratory for examination. There was about 10 c.c. of fluid. A few masses of currant jelly

like medulla were also removed and sent to the laboratory, as was a section from the cyst wall. There were no areas of whitish tissue, characteristic of fibrous osteitis. The interior of the expanded cortex was curetted with Volkmann's sharp spoon, the opening in the cortex being enlarged until about 8 cm. long and 2 cm. broad, the defect extending from the level of the epiphyseal cartilage to the insertion of the deltoid muscle. At these limits the cortex and medulla seemed to become normal. The cortex on the median side of the bone opening was then crushed in, and the wound was closed in layers without drainage: first, the periosteum, then the deep fascia, and finally the skin. The time consumed by the operation was one hour.

The Esmarch bandage and Wyeth's pins were removed after the wound had been dressed. A shoulder cap was applied, and the arm bandaged to the side.

Course.—At the first dressing, January 22, all the skin sutures were removed. There was moderate serous discharge (subcutaneous) from below the lowest suture. The rest of the wound was healed. The arm was dressed again three days later, when there was no discharge, the wound being soundly healed. The boy was sent home this day, two weeks after operation.

There have been no symptoms since operation, and in October, 1922, nearly ten years after operation, there was no evidence of recurrence and no disability (Fig. 8).

Pathologic Report (Dr. C. Y. White).—Cyst Wall: Small fragments of the inner surface and wall of the cyst showed, on histologic examination, normal bone trabeculae, faced by a narrow margin of fibrous tissue. The latter separated the cyst contents from the bony structure and consisted of typical fibrous tissue cells without other cellular infiltration. The bone fragments consisted of normal bone trabeculae without cellular infiltration other than is common to spongy bone.

Cyst Contents: The contents of the cyst showed yellowish fluid containing blood clots. Smears made from the fluid showed the following cellular contents: polymorphonuclear leukocytes, 14 per cent.; mononuclear cells, 2 per cent.; transitional cells, 1 per cent.; lymphocytes, 72 per cent.; eosinophils, 2 per cent.; neutrophilic myelocytes, 9 per cent.

No nucleated red cells, no giant cells, a few fibrin fibrillae and serum were found.

Bacterial Cultures: Anaerobic and aerobic cultures were negative. No bacteria were observed in the various smears examined. A complement fixation test for syphilis made on the fluid from the cyst gave a negative reaction.

CASE 7.—*Multiple cystic osteitis. History.*—E. J., a brunette girl, aged 19, was admitted to the Episcopal Hospital, Oct. 12, 1920. The general health had always been good. When 7 years of age (in 1908) while walking on the street, the patient fractured the left femur—apparently a spontaneous fracture. She was taken to the Chester Hospital, where she said the bone was rebroken seven times, but finally united with marked deformity. In 1914 (at 13 years of age), she sustained a fracture at the middle of the right femur. Union occurred without deformity. Dr. H. M. Armitage operated at the Chester Hospital on the left femur twice in 1915 for deformity. (He writes that both operations were merely osteotomies with straightening of the bone for malunion following repeated fracture. He saw no indication then of any cystic disease.) In 1916 she injured the upper third of the left leg (below the knee) while playing on a sliding board; but she was not confined to bed and did not wear a splint. She had had pain in the left leg until the last year. Anteroposterior bowing of the right leg had been developing gradually for years.

Examination.—The patient could walk with crutches or other support as far as a mile, progressing very slowly, crossing one leg in front of the other as indicated in the accompanying illustrations (Figs. 9 and 10).

Only the lower extremities were affected, the left femur and leg bones being much more deformed than the right. The right lower extremity from the anterosuperior spine of the ilium to the internal malleolus measured 74 cm., the left 71 cm., in straight lines, not allowing for the distortion of the limbs (Figs. 11, 12 and 13).

The left femur slightly above the middle was bowed outward, making an angle of about 135 degrees, open toward the midline of the body (Fig. 13). There

Fig. 9 (Case 7).—Appearance of legs before operation; multiple fibrocystic osteitis, showing ordinary cross-legged position in attempts at locomotion.

was no abduction possible in the left hip. The left patella was nearly a hand's breadth above the right. Owing to the deformity, the left femur was 7 cm. shorter than the right. The left leg (below the knee) was bowed inward in a long curve at an angle of 155 degrees open laterally.

The right thigh was apparently normal except for palpable thickening on the lateral border of the femur, about 10 cm. above the condyles. Abduction was possible in the right hip to about 45 degrees. There was marked knock-knee deformity on the right, the thigh and leg making an angle of 145 degrees.

The blood Wassermann reaction was negative, and there was no Bence Jones protein in the urine, which was otherwise normal, as was the blood.

Treatment.—She was put to bed with weight extension (longitudinal) applied to both legs; lateral traction to each knee, pulling the knees apart, and a band over the apex of the deformity of the left femur pulling toward the right.

During her stay in the hospital metabolic tests were made by Dr. A. A. Walkling under the supervision of Dr. C. Y. White, director of the hospital laboratories; but they gave no conclusive results.

Dec. 17, 1920, after two months in bed with extensions as above described, the position of the left leg having been slightly improved, the patient was etherized, and, after forcible abduction of the left hip until the femur was heard to crack at the site of the old fracture, a plaster-of-Paris dressing was applied from the axillae to the toes on both sides, at the same time keeping the deformities of the knees and legs under as much tension as possible in an effort at correction.

Fig. 10 (Case 7).—Same patient, with hips abducted as far as possible.

Course.—Jan. 13, 1921, the gypsum dressing was removed from the right lower extremity. She was then able to be about the ward in a wheel chair.

Feb. 5, 1921, the patient was transferred to the Orthopaedic Hospital for further treatment.

Feb. 12, 1921, under ether, a supracondylar osteotomy of the right femur was performed, subcutaneously. The deformity was almost entirely overcome. A plaster case was applied from the toes to the hip.

April 5, 1921, the paster case was removed from the left lower extremity and the pelvis.

Fig. 11. (Case 7).—Cystic first metatarsal bone of foot.

Fig. 12. (Case 7).—Deformity and cystic changes in the tibia and fibula of both legs.

April 9, 1921, under ether, resection of the left tibia was performed. An incision, 20 cm. long, was made, forming a flap with its base on the lateral side of the leg. The periosteum was incised and reflected from the entire circumference of the tibia. The tibia was soft except along the posterior border and

Fig. 13 (Case 7).—Left femur, showing deformity and cystic changes.

was easily indented by a blunt instrument. Free bleeding occurred from the cortex. The tibia was divided obliquely by the osteotome from the median border out and down to the lateral side below, the section being about 8 cm. A spurting artery from the upper fragment of the divided tibia was controlled by

crushing in the trabeculae. Points of the tibial segments were cut off, and a square notch was cut on the apposing surfaces of the tibia. Much better position was secured by approximating these sections (Fig. 14). Reduction was maintained by one Parham band. The periosteum and fascia were sutured with interrupted chromic catgut No. 1 and the skin with continuous catgut No. 0. The fibula was not divided. The time required was forty-five minutes. A plaster cast was applied from the toes to above the knee.

July, 1921, caliper braces were applied to both lower extremities, and the patient returned to her home, temporarily. She walked with crutches.

September, 1921, she wrote that she was "improving wonderfully." She walked with one crutch.

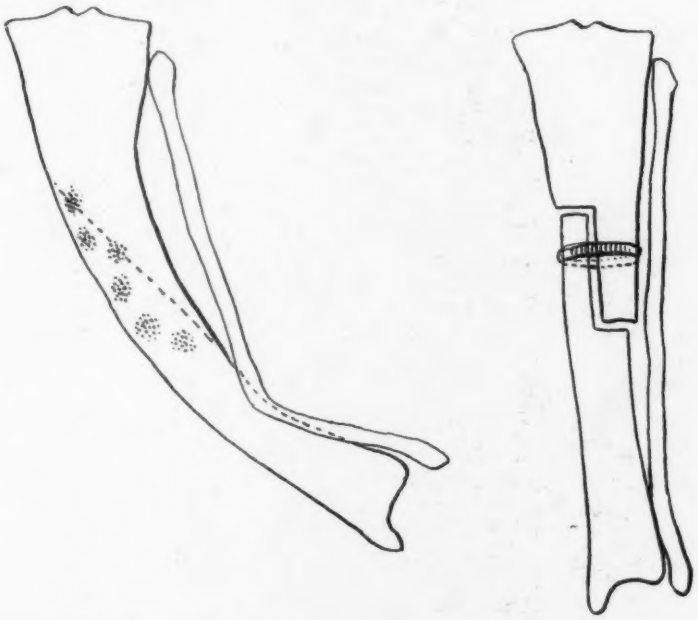

Fig. 14 (Case 7).—Diagrams, showing nature of operation on left leg.

January, 1923, she still used one crutch, and had slight "cross-legged" progression. The brace was still on the left leg. There was firm fibrous union in the left tibia. She works every day in a mill (Fig. 15).

Pathologic Report (Dr. C. Y. White).—The findings were osteitis fibrosa; no giant cells.

CASE 8.—*Multiple fibrocystic osteitis. History.*—H. R., a brunette youth, aged 18, was admitted to my service at the Orthopaedic Hospital, Oct. 21, 1916, presenting the deformities shown in the accompanying illustrations (Figs. 16, 17, 18 and 19). He had been in normal health from birth until the age of 5 years; from 5 to 7 years of age his bones seemed to soften and bend, and at 9 years of age he suffered his first fracture, from trivial injury. Since the age of 9 years he had had seven other fractures of the limbs; but there had been no fracture for the last few years, and the disease seemed quiescent.

Roentgenograms of the various bones are reproduced in Figures 20, 21 and 23.

First Operation.—In order to correct the deformity of the right leg, resection of the right tibia and fibula was performed Oct. 28, 1916. They were dovetailed together according to the diagram in Figure 24. The first dressing was made Feb. 10, 1917, three and one-half months after operation, when the gypsum case was removed. The soft parts were healed normally; but there was considerable movement in the tibia. A new case was applied.

Second Operation.—Dec. 16, 1916, operation was performed on the left femur. This bone was almost subcutaneous at the apex of the outward curve. The periosteum was detached by sharp dissection, leaving some flakes of cortical bone

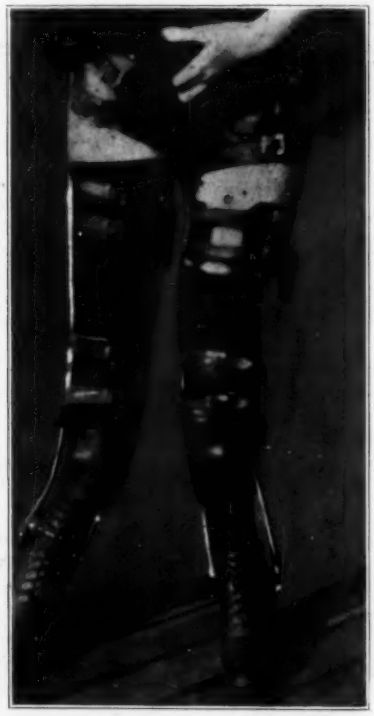

Fig. 15 (Case 7).—Results of operative correction of deformities.

attached, from the entire circumference of the femur for a longitudinal distance of 8 or 10 cm. The linea aspera jutted out as a very prominent ridge and was difficult to clear. The shaft of the femur was soft and easily dented by the periosteal elevators. It was planned to section the bone as indicated in the first diagram of Figure 25, and then to resect enough of the ends to allow the femur to become nearly straight. It was realized that a simple wedge resection would be inefficient owing to the tension of the soft parts in the adductor and flexor regions. A Hey's saw and osteotomes were used for making the section; but the femur broke across (second diagram of Fig. 25) after section *A* was completed. This was, therefore, removed, and section *B* was cut separately. When the femur was straightened, the condition was that shown in the third diagram of Figure 25. The distal half of fragment *A* was replaced in the gap, and the fragments were held together by two encircling pieces of phosphor bronze

Fig. 16 (Case 8).—Right lateral view of patient with extreme deformity of extremities from multiple fibrocystic osteitis.

Fig. 17 (Case 8).—Left lateral view.

wire. These were not sufficient to prevent slight motion, but did prevent gross displacement of the fragments, and thus facilitated closure of the soft parts and application of the plaster-of-Paris dressing, which included the pelvis, with the hip flexed to 120 degrees and the knee to 30 degress, permitting the sole of the foot to lie flat on the bed. The first dressing was done Feb. 10, 1917, two months after operation. The wound was healed except for one point of granulation. A new plaster-of-Paris dressing was applied (not including the pelvis), with the hip extended and the knee at an angle of 160 degrees. There appeared to be fibrous union in the femur. March 7, 1917, preparatory to a second operation on the left leg (compare description of the fourth operation), the case was removed from the left thigh and leg, and a new one was applied including

Fig. 18 (Case 8).—Anterior view.

the left hip and the thigh to just above the knee. Union was not yet firm in the left femur.

Third Operation.—Feb. 17, 1916, this was performed on the right femur. This bone was bowed outward at right angles below the trochanters and had a cyst, 4 cm. in diameter, at the apex of the deformity. The neck of the femur made an angle of about 80 degrees with the shaft (coxa vara). Before the left femur was straightened, the right thigh crossed the left; but since the operation on the left femur, the thighs could be abducted just far enough to put the hand between them. The right thigh still was so markedly adducted below the deformity that it made an angle of 45 degrees with the long axis of the body (Fig. 26).

At the operation, it was found that the periosteum stripped rather easily from the rough, porous and soft bone. The periosteal elevator easily perforated the cortex into the largest cyst, and about 4 c.c. of clear fluid was discharged. The periosteum being reflected, the femur was split, by osteotome, longitudinally in the frontal plane of the patient's body for a distance of 12 cm., and the distal end of this section was connected by saw with the extensor surface of the femur, and the proximal end with the flexor surface of the femur. In attempting to

Fig. 19 (Case 8).—Appearance of patient with legs extended.

straighten the femur by sliding these ends past each other, the distal fragment was fractured nearly transversely, a little below the cyst. The loose fragment, including the cyst, was removed (Fig. 22). The ends were then overlapped and encircled by two Parham steel bands, as indicated in Figure 26. After closure of the wound in layers, a plaster-of-Paris dressing was applied from the toes to the axillae, with the right femur abducted 45 degrees. The dressing, two and one-half months after operation (April 25, 1917), showed the soft parts healed and union firm.

Fourth Operation.—March 10, 1917, an operation was performed on the left leg. The apex of the deformity (convex medially) was exposed, and the periosteum stripped from the tibia, leaving flakes of bone attached. The bone was cystic in places and quite soft. The tibia was split for 10 cm. in the coronal plane, the section emerging on the extensor surface of the tibia at its proximal end and on the flexor (calf) surface at its distal end. The fibula was fractured

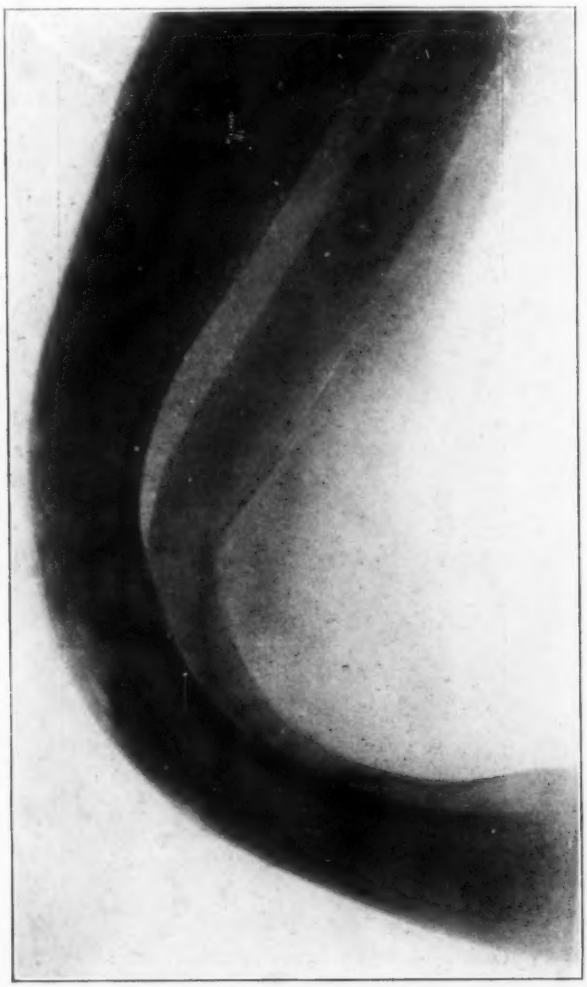

Fig. 20 (Case 8).—Right tibia and fibula.

purposely by momentarily increasing the deformity of the leg, and the ends of the fragments of the tibia were thus exposed. An artery in the medulla of the proximal fragment ceased to spurt after the application of gauze pressure for a few minutes. The tibial fragments were then overlapped until the axial deformity was nearly overcome; whereupon the superfluous bone was sawed off, causing the fragments to fit together well (Fig. 27*A* and *B*). These were encircled by two

Parham bands. The lower band cut into the soft bone for about 1 cm.; but the upper band held firm on the surface of the cortex. After closing the wound, plaster of Paris was applied to the leg and incorporated with the gypsum dressing already on the thigh.

Fifth Operation.—This proved fatal to the patient. It was undertaken, April 28, 1917, on the right leg, which had been the seat of the first operation, just

Fig. 21 (Case 8).—Left tibia and fibula.

six months previously. It was desired to complete the correction of the deformity of this leg. No Esmarch bandage for temporary hemostasis was employed, and none had been employed at any of the previous operations. No unusual hemorrhage had been encountered at any of the operations, the spurting artery from the medulla, noted in the account of the fourth operation, was the only

Fig. 22 (Case 8).—Portion of right femur resected for cystic disease with deformity, Feb. 18, 1917.

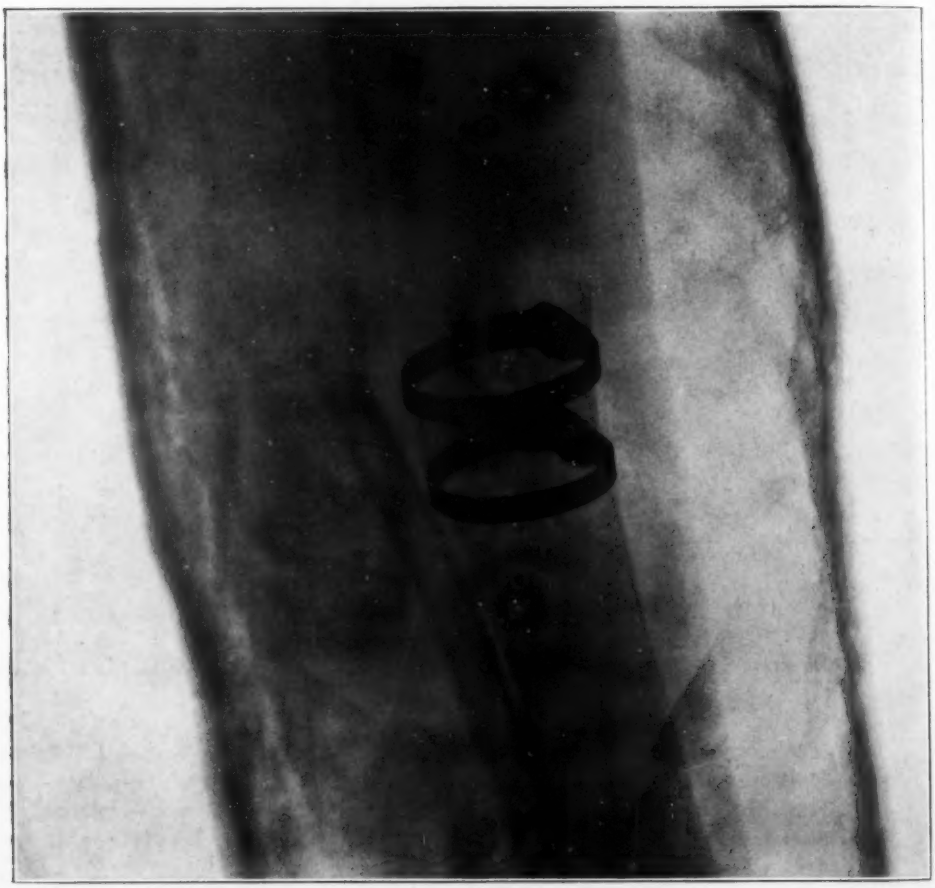

Fig 23 (Case 8).—Left leg, April 17, 1917, five weeks after operation; fixation by Parham bands; compare with Figure 21.

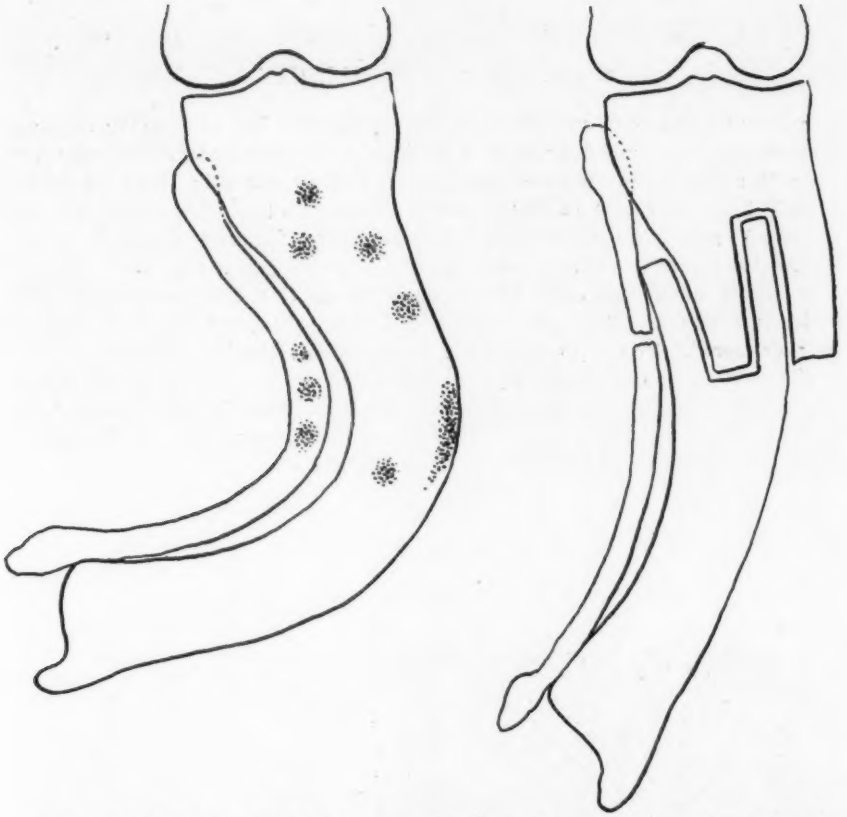

Fig. 24 (Case 8).—Right leg: resection of tibia, with dovetail joint for fixation. This illustration and Figures 25, 26, 27 and 28 are diagrams of the numerous operations employed to correct deformities.

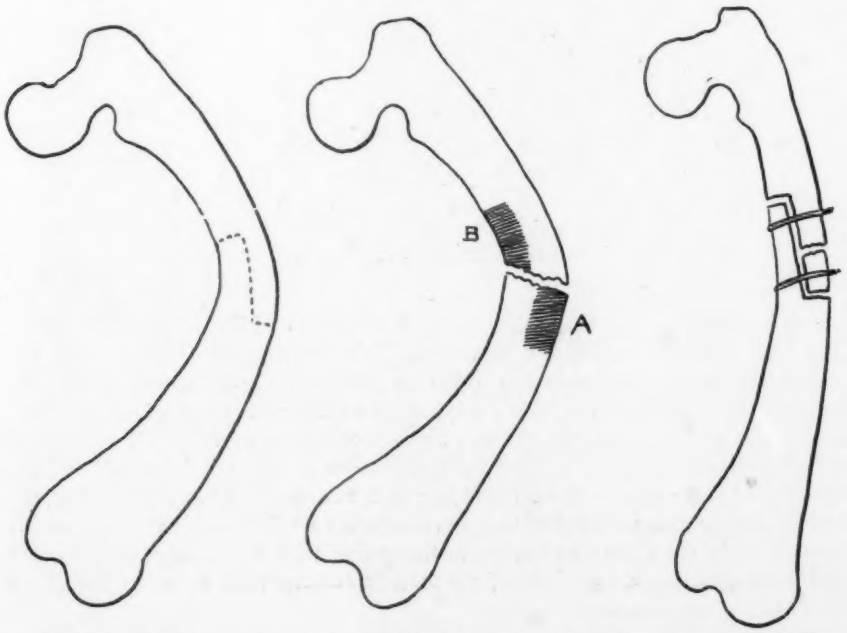

Fig. 25.—Left femur: resection, with fixation by phosphor bronze encircling wire.

bleeding that had been considered worthy of record. But in this, the fifth and last, operation in making the skin incision the internal saphenous vein was cut, and, owing to its being embedded in scar tissue against the periosteum, quite a little blood was lost before it was clamped and tied. This was the only untoward feature of the operation. There was firm fibrous union at the site of the former resection of the tibia (Fig. 24); but the fragments could be moved just enough upon each other by the hands to make the motion visible. The periosteum was reflected; the bone divided along the former sections, and the ends delivered. The fragments were sawed as indicated in Figure *27C*, and it was necessary to resect the fibula to allow apposition of the ends of the tibia in the correct axis. The tibial fragments were encircled by one Parham band, which gave perfectly firm fixation (though no indiscrete attempts were made to try its strength). To keep the band from slipping down the conical surface of

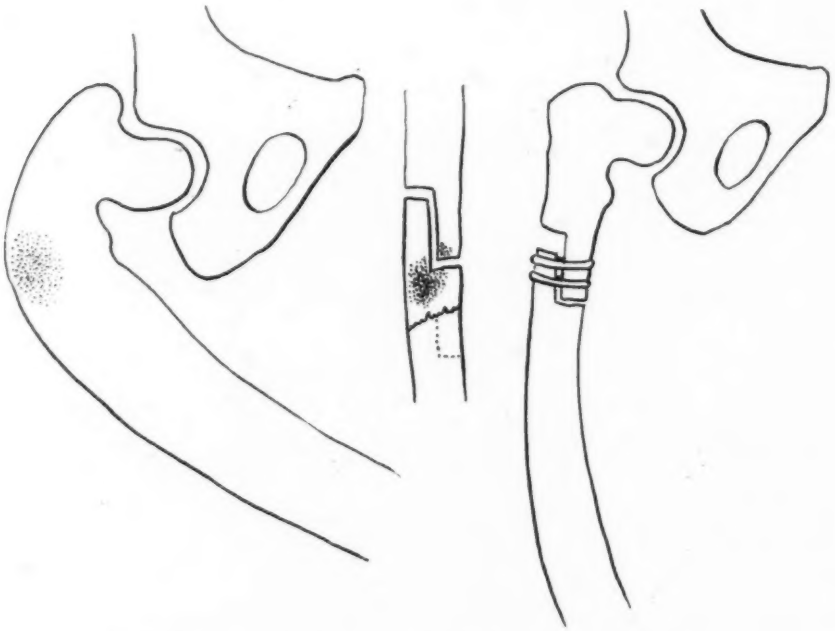

Fig. 26.—Right femur: resection, with fixation by Parham bands.

the tibia, a groove was cut in the crest of the bone, in which the band was fitted. The wound was closed, and a plaster-of-Paris dressing was applied from the toes to the groin. The operation lasted an hour, and the patient was distinctly shocked. But as his pulse had always risen to 160 at the end of the previous operations, no undue alarm was felt at the conclusion of this. He was given stimulation, put to bed with local heat, a hot enema was administered, and the foot of the bed raised; but he failed to react, and died about an hour after the end of the operation. Failure to appreciate the cumulative effect of five major operations in the course of six months, perhaps with the little extra bleeding and delay occasioned by injury of the long saphenous vein, seems to have been the cause of the fatal termination.

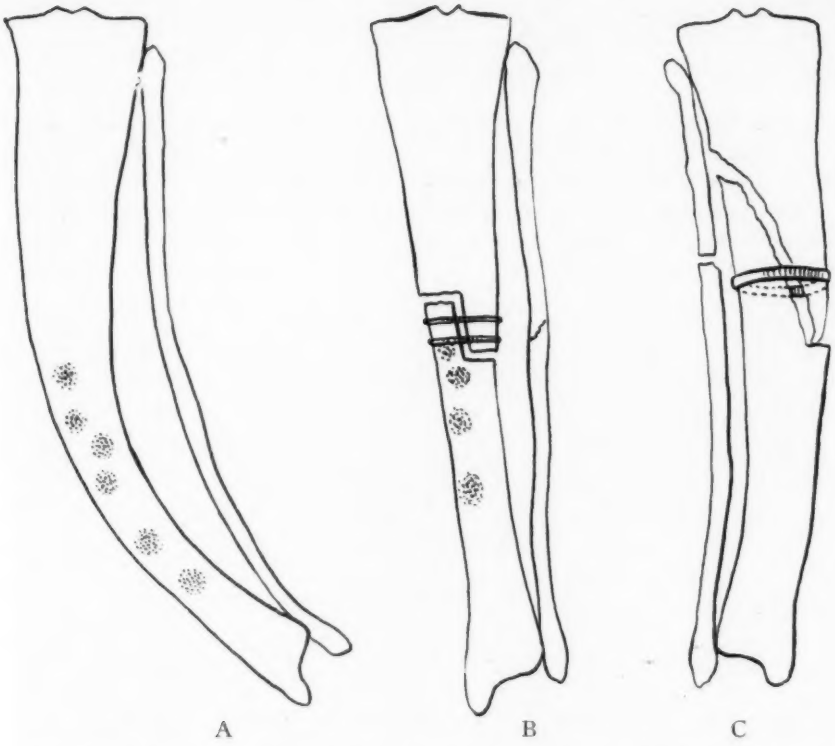

Fig. 27.—*A*, left leg; *B*, resection of tibia, with fixation by Parham bands; *C*, right leg, second resection, with fixation by one Parham band.

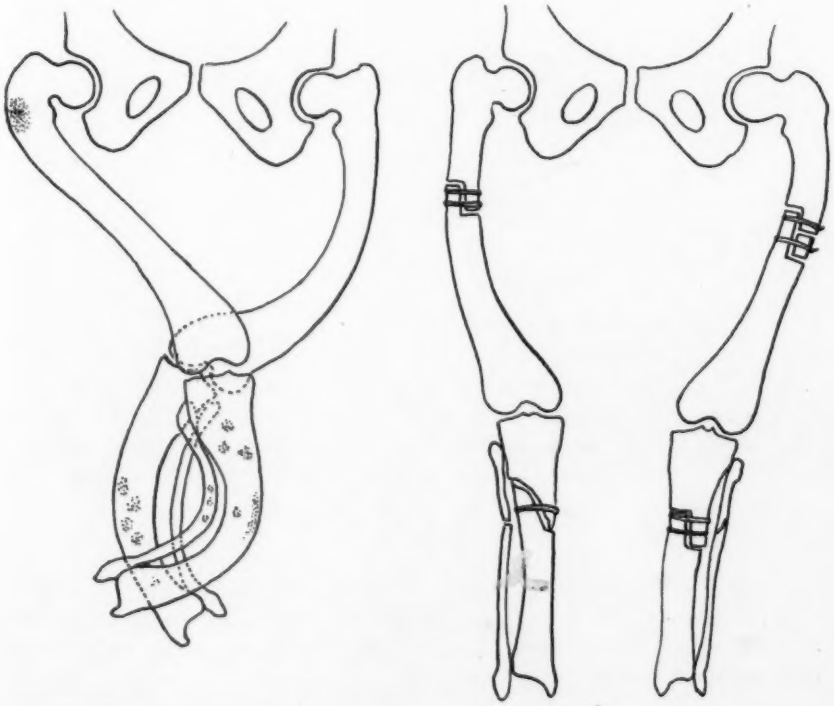

Fig. 28.—Lower limbs before any operations and after last (fifth) operation.

Pathologic Report (Dr. C. Y. White).—The findings revealed typical osteitis fibrosa. Between spicules of bone, the sections examined were made up of loose fibrous tissue, with fully formed capillaries and larger blood vessels. The bone stained more deeply than the normal in areas, and were more cellular than normal—apparently undergoing absorption. No giant cells were seen.

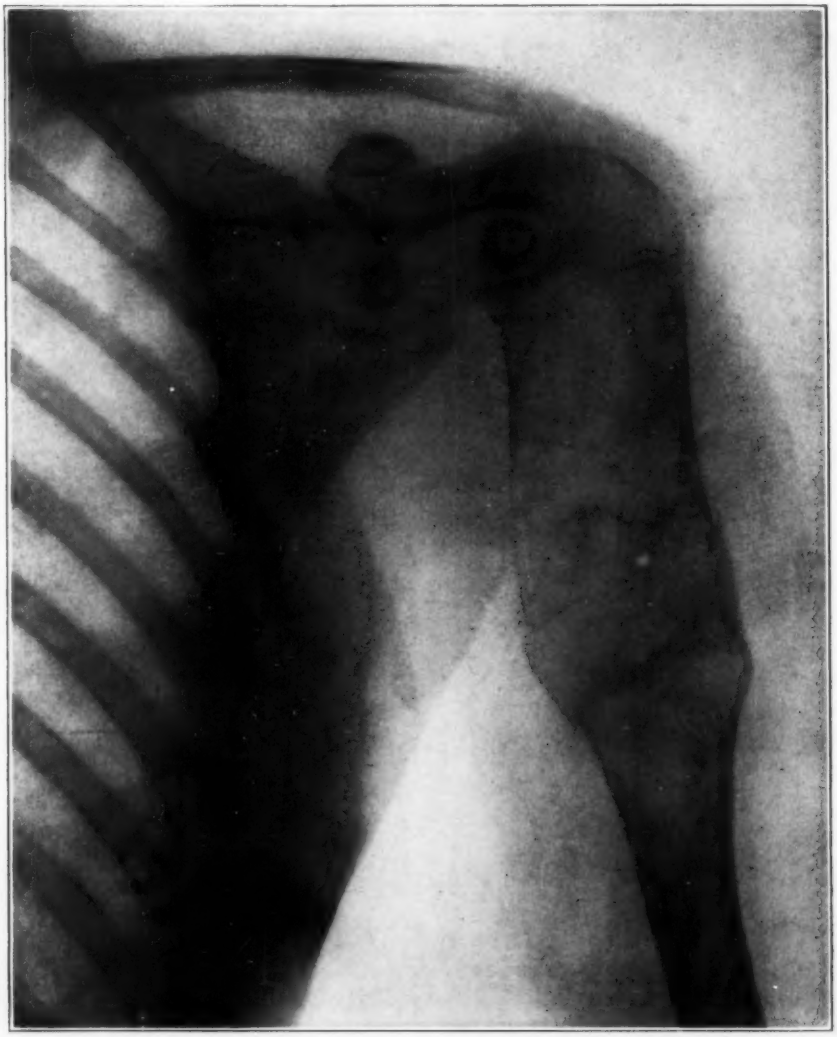

Fig. 29 (Case 9).—Cystic osteitis of the left humerus.

Metabolic Studies.—While the patient was in the Orthopaedic Hospital metabolic studies, made by Dr. E. P. Corson White, indicated an excessive loss in the excreta of calcium and phosphorus. Dr. White believed that the disease was of the osteomalacia type.

CASE 9.—*Myeloma (giant cell sarcoma) of the humerus. History.*—S. B., a brunette girl, aged 13, was admitted to the Episcopal Hospital Nov. 11, 1920. In November, 1919, a tonsillectomy had been performed.

In 1916 she fell and bruised the left arm. It became swollen and her mother rubbed it with liniment. The swelling soon subsided and she had no further trouble; but she said that "if any one frightened her after this her arm became numb and she was unable to move it at the shoulder." She had injured the arm several times since 1916, and each time it became swollen; but the swelling always subsided in a few days.

Aug. 1, 1917, she was treated in the surgical dispensary of this hospital for an injury of her left arm, for which liniment was prescribed.

Nov. 8, 1920, she fell downstairs, striking her left shoulder, and was unable to lift it. She came to the surgical dispensary where she was seen by Dr. W. R.

Fig. 30 (Case 9).—Diagram showing nature of operation employed.

Brown, Jr., who, after having the arm roentgenographed, referred her to the service of one of us (A. P. C. A.).

Examination.—She was a well developed stout Jewish girl, normal except for the condition of the left arm, which was somewhat swollen from the shoulder almost to the elbow and felt slightly hot to the touch. The bone was thickened. There were a false point of motion and crepitus about the middle of the humerus. She was unable to use the arm.

Roentgen-Ray Report (Dr. R. S. Bromer, Fig. 29): "Upper half of left humerus shows marked cystic formation. This does not resemble one huge bone cyst but has the typical appearance of cystic osteitis. There is a pathologic fracture of the shaft of the humerus." Dr. Ashhurst: "Roentgen ray shows cystic expansion of upper part of humerus as far down as insertion of deltoid to more than twice its normal diameter. The expansion is not abrupt at the

lower limit of disease, and extends up to the epiphyseal cartilage of the head, which is still visible. There are numerous trabeculae in the cystic area and a transverse fracture through the middle of the cyst, without displacement."

Operation.—Nov. 16, 1920, Esmarch anemia was secured by the use of a rubber bandage around the shoulder, held in place with Wyeth's pins, as in cases of amputation at the shoulder joint (compare operation in Case 6).

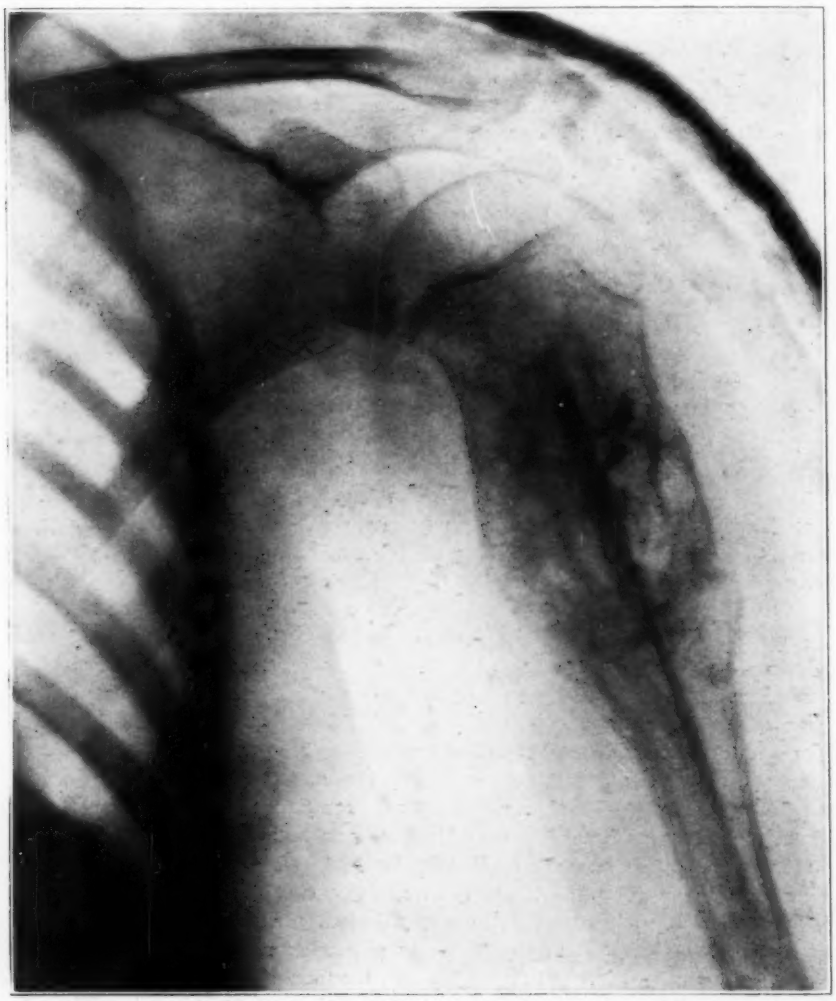

Fig. 31 (Case 9).—Left humerus, fifteen weeks after operation.

An incision was made, 20 cm. in length, from the greater tuberosity of the humerus downward, just laterad to the biceps muscle. When the periosteum was reflected from the bony promience, a subperiosteal fracture was disclosed at the middle of the bony swelling. The cortex of the humerus was almost deficient at this point and was easily perforated by a periosteal elevator. When

opened it was found to be only 1 mm. thick. Inside the cyst were several bony trabeculae, incompletely dividing it into the different cavities. The contents of the cyst consisted mostly of yellow, slightly turbid fluid, with a little currant jelly like tissue. A culture was taken, and this, as well as specimens of the

Fig. 32 (Case 9).—Left humerus, nine months after operation.

bone marrow and cortex, was sent to the laboratory. The marrow cavity was thoroughly cleared out with the curet as far as the epiphyseal cartilage above, and downward to the point where the cortex and marrow appeared normal; although even here the marrow appeared grayish and congested.

An aperiosteal transplant was then cut from the subcutaneous surface of the left tibia, 15 by 1.25 by 1.25 cm., and was fitted snugly into the interior of the cyst, a slot being cut in the shaft distal to the cyst, in order to admit the transplant without disturbing the fragments of the humerus (Fig. 30). The cyst walls were then crushed in, considerably diminishing the size of the cavity. After complete closure of the soft parts in layers, the Esmarch bandage was removed, and the entire upper extremity and thorax encased in plaster of Paris, with the shoulder abducted about 45 degrees.

Laboratory Report (Dr. C. Y. White).—The fluid from the tumor was serosanguineous. An unstained film showed nothing pathologic. A stained film showed the usual red and white cell elements, with slight excess of lymphocytes. Culture of the fluid showed it to be sterile.

Microscopic: Sections of fragments of tissue submitted were made up of: (1) fibrous tissue similar in every respect to scar tissue which bordered a small section of bone; (2) larger spicules of bone undergoing absorption. Surrounding the bony spicules were areas of hemorrhage. In their immediate vicinity were a few giant cells (epulis type).

Diagnosis: This was fibrocystic osteitis with giant cells.

Course.—Nov. 23, 1920, a slight continuous fever had been present since operation, from 99 to 101 F. The temperature was normal today. The patient sat up in a chair all day.

November 26, the leg wound was dressed. The incision was healed.

December 11, she was put back to bed because af fever (101 F.), and a purgative was given.

December 17, the temperature was normal. The patient was out of bed.

December 22, she walked about the ward.

Jan. 7, 1921, seven weeks after operation, the case was removed from the arm and chest, and the first dressing was made. All skin sutures were infected. No deep infection was apparent. Union was firm in the humerus. A new case was applied to the arm and chest, and the patient was discharged from the ward the next day.

March 4, 1921: The arm had been dressed weekly since the last note through a window in the cast. Healing was complete, except for a small granulating area at the upper end of the incision. The case was removed today and a shoulder cap of gypsum was applied. There was firm union and no deformity. The roentgen ray (Fig. 31) revealed the bone much denser and less expansion. The transplant was intact; there was no deformity.

Aug. 1, 1921, a sinus had persisted. Recently a small sequestrum was discharged spontaneously. As there was no disability and the girl was able to use her arm normally, the parents declined any radical treatment for cure of the sinus. A roentgenogram (Fig. 32) showed the transplant absorbed and the humerus to be of nearly normal density.

June, 1922: The sinus healed in September, 1921, and had remained closed. There was no disability.

CASE 10.—*Myeloma (giant cell sarcoma) of the lumbar vertebrae.*[13] *History.*—O. N. a brunette boy, aged 14 years, was admitted to the Orthopaedic Hospital, Dec. 28, 1913. Oct. 13, 1913, he had strained his back by lifting one end of a 300 pound elevator weight. That night he suffered severe pain in

13. This case has already been the subject of a report by Dr. G. B. Denit (Ann. Surg. **62**:636, 1915).

the back, radiating from the lumbar region to the right hip. In several days the pain ceased. One month later, while playing at school, he was knocked down and fell against a stone wall, striking the right lumbar region. This caused so much pain that he was taken home and put to bed. After being in bed for a time he got about on crutches; but, Dec. 1, 1913, he was taken to a hospital in his home city and treated for rheumatic arthritis of the right hip.

Fig. 33 (Case 10).—Roentgenogram, one month after operation, of a myeloma involving the right side of the lumbar vertebrae.

He was transferred thence to the Orthopaedic Hospital, four weeks later, with the diagnosis of "progressive muscular atrophy."

Examination.—On admission he was suffering with severe pain in the right lower quadrant of the abdomen and in the right hip. This pain was paroxysmal in character, lasting an hour or more at a time. He was a fairly well

nourished boy and about the average size for his age. In the right lumbar region was a large mass, semisolid in consistency, occupying the entire right lumbar region and the right lower quadrant of the abdomen. This mass was extremely tender on palpation. The knee jerks were increased, and there was bilateral ankle clonus.

Fig. 34 (Case 10).—Appearance of vertebrae, five months after operation.

Two days after admission, the temperature was 101.6 F., having varied between 99 and 100 F. ever since admission. The leukocytes numbered 9,000; the urine was negative. The boy had had several severe attacks of pain in the right loin, writhing, moaning and jerking the limbs during these attacks. He lay either prone, or on the right side, always with the right hip flexed. Today there was no ankle clonus, and the knee jerks were absent on both sides. He complained of much referred pain in the right knee. The mass in the loin

remained about the same as on admission, but anteriorly it could be more clearly outlined, extending from the costal border to below the level of the umbilicus. The right iliac fossa was free, and the lower edge of the mass was easily outlined above it. The mass did not move in respiration and was quite fixed on palpation and moderately tender. It was continuous with the liver dulness. Roentgen-ray examination of the spine was negative.

Fig. 35 (Case 10).—Vertebrae, seventeen months after operation.

The preoperative diagnosis was: (1) probably a cold abscess, though it was considered very unusual for such a lesion to be outlined so clearly and so firm as this tumor, or for an abscess of this size to form within so short a time (two and one-half months) and without demonstrable lesion of the spine; (2) perhaps a hypernephroma or sarcoma; but it was thought that the spinal cord symptoms favored the diagnosis of cold abscess.

Operation.—Under ether anesthesia operation was performed Dec. 30, 1913. After incising the lumbar fascia through an 8 cm. incision in the right loin, a well encapsulated bluish tumor was exposed, easily outlined below and in front. It was shaped like the lower pole of the kidney, but was much larger, and not quite so firm in consistency. When the capsule was punctured, there was a gush of semiclotted blood, about 500 c.c., mostly fluid, being evacuated. The lumbar and sacro-iliac joints could be palpated apparently free from all muscular attachments; but there was no area palpable which seemed to be devoid of periosteum. Two of the transverse processes of the lumbar vertebrae felt bare, and the erector spinae muscle seemed destroyed. Fragments of tissue (muscle? tumor? angiosarcoma?) were saved for examination. The rather profuse hemorrhage stopped after complete evacuation of the diseased tissue; the cavity was packed with iodoform gauze around a

Fig. 36 (Case 10).—Low power photomicrograph of tissue secured at operation: *A*, blood clot; *B*, tumor tissue.

rubber tube, and the wound was closed in layers. The gauze and tube were removed during the first week.

Pathologic Report.—One specimen of the tumor was examined by Dr. E. P. Corson White, pathologist to the Orthopaedic Hospital, and another section by Dr. C. Y. White, director of the laboratories of the Episcopal Hospital. The report from both laboratories was that the tumor was a "giant cell sarcoma" (Figs. 36 and 37).

Course.—As the swelling in the loin began to reappear during the third week after operation, treatment with Coley's fluid (mixed toxins of streptococcus and *Bacillus prodigiosus*, supplied by Dr. Martha Tracey) was begun. A roentgenogram taken one month after operation (Fig. 33) showed a moderately dense

and irregular shadow in the right loin, extending from the third, fourth and fifth lumbar vertebrae to the crest of the ilium.

Jan. 22, 1914, three weeks after operation, the first injection of Coley's fluid (1/10 minim) was given into the buttocks. Seventeen weeks after the initial dose, the amount administered had been gradually increased until 42 minims were being given. At no time during these weeks were the reactions severe. After an interval of three weeks, during which no Coley's fluid was given, injections were resumed, the first dose being 42 minims, the amount being gradually increased until 48 minims had been reached. Again an interval of rest for three weeks was allowed, after which the injections were resumed, beginning with a dose of 48 minims. From this time on the reactions were rather severe, the temperature often rising to above 102 F. and at times reaching 103.2 F. Chills accompanied the temperature, and there was always severe pain at the site of

Fig. 37 (Case 10).—High power photomicrograph of tissue secured at operation: *A*, giant cell; *B*, blood pigment; *C*, giant cell.

the needle puncture and often marked skin reactions. The highest dose reached was 56 minims. At first, as already stated, the injections were made in the buttocks, first on one side, then on the other, gradually approaching the site of the tumor, until finally the injections were given directly into the tumor in the loin. These injections were continued over a period of eight months, with only two periods of rest, of three weeks each. During these intervals the patient's general condition improved a great deal. The injections were never given on two successive days, but two or three times weekly. Morphin, codein or acetylsalicylic acid (aspirin) was frequently required for pain, but though tonics were required, the lad's state never became so bad as to require stimulation. The reactions on the whole were very mild, but occasionally very

severe. Dr. Denit, in his original report, states that in a letter received from Dr. Martha Tracey, Philadelphia, whose preparation of Dr. Coley's toxins was used, she stated that the highest dosage employed by Dr. Coley was 26 minims, though she knew of one case in which 30 minims was given at a dose. Dr. Denit believed that the dosage reached in the case now reported, 56 minims, was the highest yet reported.

The tumor, which, after the operation wound had healed, had begun to increase in size (Fig. 34), remained about the same in consistency until the end of September, at which time it began to become softer and to increase markedly in size. Several days later the mass broke down and discharged through the scar of operation in the loin. From this time the mass diminished

Fig. 38 (Case 11).—Myeloma involving the head of the tibia.

in size and became harder in consistency until at the time the injections were discontinued, it had the consistency of a bony tumor. This tumor seemed to be adjacent to the fourth and fifth lumbar vertebrae in the right loin.

The patient was discharged, Nov. 10, 1914. He had been up and about the ward almost daily since early in February, about two weeks after the administration of Coley's fluid was begun, when the pain had almost subsided. On discharge, no tumor could be palpated through the abdominal wall, and the mass in the loin had decreased markedly in size and assumed the form of a bony tumor. At this time the patient's weight was 76½ pounds (35.4 kg.). Six months later it was 103 pounds (47 kg.). He appeared to be in perfect health, and there was no increase in the size of the bony tumor, which appeared as a dense bony mass in the roentgenogram (Fig. 35).

In August, 1920, nearly seven years after operation, he was reported as being in good health. He had served through the war without disability, but he had had the misfortunte to lose an eye.

CASE 11.—*Myeloma of head of left tibia. History.*—Lieut. J. D., U.S.A., brunette, aged 23, was under my care at the Walter Reed General Hospital, Washington, D. C., in April, 1919. Nine years previously, when 14 years of

Fig. 39 (Case 11).—Myeloma involving the head of the tibia, duration nine years.

age, he received a violent blow below the left knee from a heavy piece of wood. He was in so much pain that he was unable to walk for an hour and a half. He then hobbled home. Within a few months, a bony lump made its appearance, but caused no disability. It grew slowly larger for several years; but he was convinced that it had grown no larger during the last five years,

as he was able still to wear the same pair of tight fitting knee breeches that he had made five years previously.

Examination.—There was a visible mass bulging on both sides of the crest of the tibia at its head; the crest also bulged forward. The appearance was similar to that of the normal knee joint, but palpation demonstrated the real knee joint, which looked like the thigh, entirely above the area where the bony tumor existed in the head of the tibia (Fig. 38). Roentgenograms, made by Major Selby, showed the typical trabeculated appearance of a myeloma (Fig. 39). On account of the patient's age, which made the presence of sarcomatous changes more likely than in a child, and because of the persistence

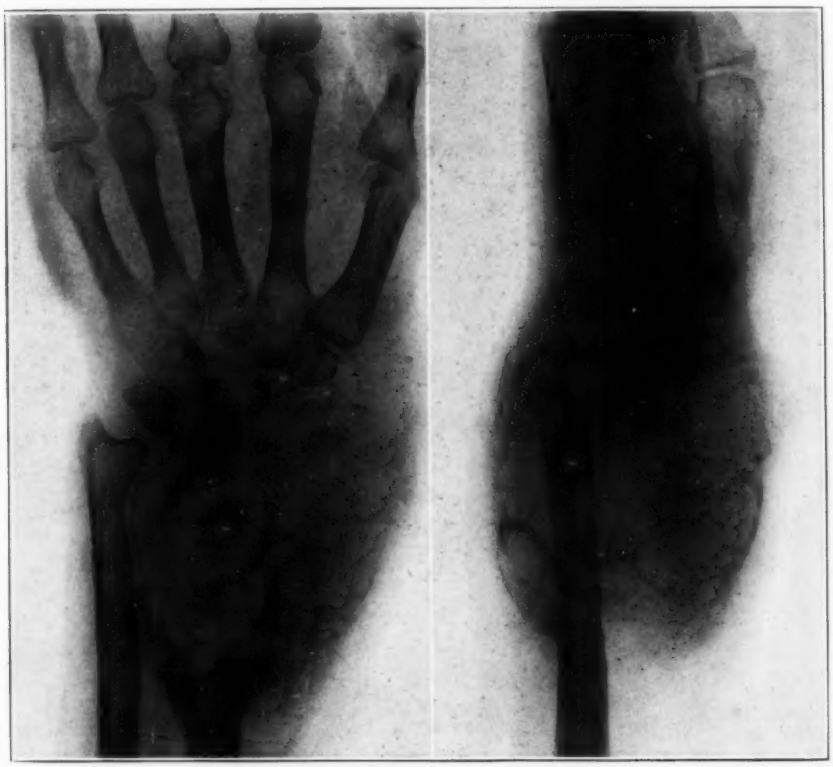

Fig. 40 (Case 12).—Myeloma involving the lower end of the radius, just before first operation, Feb. 20, 1913.

of the tumor, operation was urged, that the true diagnosis might be ascertained and an intelligent prognosis furnished to his commanding officer, the patient being desirous of remaining permanently in the army.

Operation.—April 14, 1919, under Esmarch anemia, the cortex was removed over the median and lateral aspects of the head of the tibia, leaving the crest and tuberosity intact. The lateral surface was bosselated, and the periosteum was very adherent. There was no fibrous lining to the cyst, which was multilocular and contained some yellow fluid, tissue resembling currant jelly and

softened bone. The interior was cleared out by gouge and curet until normal bone was reached in all directions. The sides and anterior wall, except the crest, were then crushed in, partially filling the cavity. The periosteum, fascia and skin were closed in separate layers before removal of the Esmarch bandage, and a plaster-of-Paris dressing was applied.

Pathologic Report.—This appears to refer only to a specimen of the cortex removed: "Absence of lacunae and osteoblasts, no evidence of inflammatory reaction; bone sclerosis."

Fig. 41 (Case 12).—Radius, seven months after first operation.

Course.—In July, 1919, after my return to Philadelphia, he wrote me that he was walking all he wanted with crutches but with no disability.

In September, 1919, he visited my office, still using a cane, as his leg felt a little weak. He was not able to run. The right tibia measured 29.5 cm. below the knee; the left, 32 cm. Recent roentgenograms showed normal bone throughout the head of the tibia.

In September, 1921, he wrote that only a small lump of bone remained (this was the protruding tibial crest, not crushed in at operation) and that he had

no disability whatever, except that sometimes in damp weather or after a very strenuous day's work, he was conscious of a little "rheumatism" in the leg.

In March, 1923, he confirmed the last report in person.

CASE 12.—*Myeloma of radius; recurrence after curettage; excision and transplantation of bone. History.*—S. M., a brunette woman, aged 22, applied to Dr. Harte's service at the Orthopaedic Hospital, Feb. 20, 1913, with a cystic swelling of the wrist, which at first sight was mistaken for tuberculosis of the tendon sheaths. The swelling had gradually developed during the preceding year (since February, 1912) from no well ascertained cause. A roentgenogram (Fig. 40) showed the lower end of the radius destroyed by a cyst, which, however, left the wrist joint and the overlying soft parts intact. March 5, 1913, Dr. Harte opened and curetted the cyst, whose walls were very thin, there being scarcely any trace of bony tissue discernible.[14] A transplant was cut from the tibia and inserted in the interior of the cyst to maintain the form of the bone and in the hope of stimulating osteogenesis. For months the local condition continued

Fig. 42 (Case 12).—Radius, fifteen months after first operation, showing recurrence just before second operation, June, 1914.

to improve. The tumor became smaller and its walls firmer; it lost its cystic feel, but the forearm remained abnormally large and caused the patient considerable annoyance. A roentgenogram taken Sept. 24, 1913, six months after operation, showed that the transplant was being absorbed (Fig. 41). Early in 1914 the tumor again began to soften (Figs. 42 and 43); the local heat and discomfort increased, until finally in June, 1914, the girl consented to another operation. She was referred to my service at the Episcopal Hospital.

Operation.—June 27, 1914, I excised the entire lower end of the radius, including its periosteum and articular surface. It was found that the extensor tendons of the thumb passed through a canal in the tumor, and while one of the tendons could be pulled out of this canal after being divided, the other could not be extracted and about 2 inches of it had to be removed with the tumor. Moreover, the tumor so overhung the carpal bones, although it had not broken

14. Dr. Paul A. Lewis, then of the Pennsylvania Hospital, reported that the growth was a "giant cell sarcoma."

through the articular surface of the radius, that it was impossible during the operation to outline its limits precisely, and the scaphoid and semilunar bones were inadvertently removed along with the tumor. A transplant, with periosteum attached, was then cut from the tibia (5 inches long, and of the thickness and shape of the lower end of the radius) and inserted in the large wound caused by removal of the tumor. This transplant was fixed to the shaft of the

Fig. 43 (Case 12).—Myeloma of radius, just before the second operation, June, 1914.

radius by a steel plate; the plate was screwed to the radius, but as the screws fixing it to the transplant twisted loose when half inserted, it was necessary to fix the plate to the transplant by a wire loop (Fig. 44). In Figures 45 and 46, the operative specimen is illustrated. Healing was uneventful. A splint was worn for three months after the operation. At that time the transplant was firmly united to the shaft of the radius; there was rotation in the forearm through an arc of more than 90 degrees; there was flexion in the wrist to 140

degrees, and extension to a straight line (180 degrees). The patient could make a good fist, and had fair power of grasping, which was rapidly improving. Figure 47 is from photographs made three months after operation.

Course.—In December, 1921, seven and one-half years after operation, there was normal strength, and the patient found the hand perfectly useful in her trade of cigaret maker.

Fig. 44 (Case 12).—After operation, June, 1914.

Feb. 12, 1922, she struck the dorsal surface of the right wrist against a table. It pained her all night, but after that she had no symptoms until February 21, when she began to have pain in the thumb and index finger. Examination, February 24, showed tenderness and slight warmth and possible false motion about the middle of the transplant, about 5 cm. above the wrist joint. The roentgen ray revealed fracture of the transplant through the site of the most distal screw holding the plate.

Second Operation.—March 11, 1922, operation was performed to remove the bone plate and the screws, and to investigate the site of fracture (Orthopaedic Hospital). Incision was made, excising the scar of the operation of June, 1914, about 15 cm. long, from the base of the thumb metacarpal upward. This was deepened to bone in front of the flexor carpi radialis. The fascia and muscles surrounding the steel plate inserted eight years previously were stained black. A fracture was found, nearly transverse and subperiosteal, involving the transplant at the site of the distal screw (Fig. 48). This screw had twisted off while

Fig. 45 (Case 12).—Roentgenogram of specimen removed at operation.

being inserted at the previous operation, and while buried in the bone did not project into the corresponding screw hole of the plate. The most proximal screw (in the original shaft of the radius) was still firmly fixed in the bone, and had to be unscrewed almost to its tip before removal. The second screw (also in the original shaft of radius) was loose, as was the third screw (in the proximal end of the transplant); but the screw at the site of the recent fracture was so densely embedded in the bone of the transplant that it could be removed only by gouging out the bone all around it. The distal encircling wire was easily

broken; but the proximal wire (Fig. 48) had to be cut. In both, the twisted ends were intact. The fracture occurred through bone, apparently dead, white, avascular and very dense, but perfectly continuous with the surrounding well vascularized bone of the transplant, which extended to within less than 1 cm. of each side of the fracture. The plate, screws and wires were removed. The junction of the transplant with the shaft could not be identified. The projecting extensor surface of the distal end of the transplant at the wrist was cut down level with the surrounding bone, and the fragments of bone thus secured were placed between the fractured ends of the transplant, after each fractured end had been gouged out until vascular bone was reached. Specimens of dead and living bone (in one piece) were saved for laboratory examination.[15] The extensor tendons of the thumb exposed above the wrist were normal but adherent to each other, and with some scar tissue around them. (Compare description of previous operation.) The wound was closed in layers with

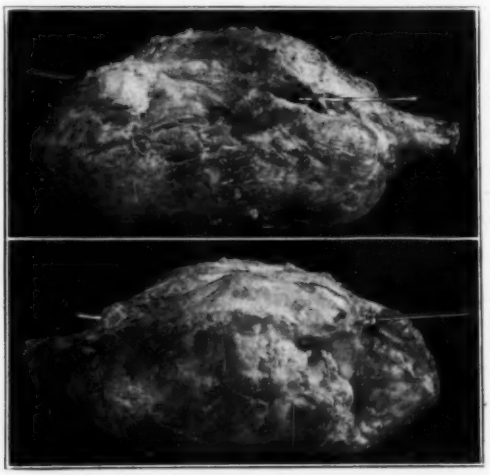

Fig. 46 (Case 12).—Specimen removed at operation: above, the extensor surface of the specimen, with a probe passed through the canal occupied by the extensor tendons of the thumb; below, appearance of flexor surface, the ulnar aspect resting on the table.

chromic catgut, and an internal angular splint was applied. Figure 49 is from a roentgenogram made four weeks after this operation.

In December, 1922, nine months after the last operation, there was not yet bony union in the fractured transplant, but the patient had been at her usual work for some months.

In February, 1923, for continued nonunion, an aperiosteal bone transplant from the tibia was inlaid across the site of fracture. Firm union was then secured. Pathologic examination of the larger pieces of bone removed from the site of fracture at this time (Dr. E. P. Corson White) showed no evidence of tumor, only sclerosed bone.

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15. Examination by Dr. E. P. Corson White, pathologist to the Orthopaedic Hospital, convinced her there was dead bone present, and also areas of "giant cell sarcoma."

Pathologic Report (Dr. C. Y. White).—On section of tissue received from Dr. E. P. Corson White, removed at operation in 1913 (Figs. 50 and 51), the report was that the general structure of the tumor mass was peculiar in that it consisted almost entirely of "epulis" giant cells, with nuclei varying in number from four or five to twenty or thirty. Between these giant cells were a few large cells with vesicular nuclei, a few small lymphocytes, a very few blood vessels (fully formed), and elsewhere blood which seemed to be either hemorrhage or blood which was coursing in spaces between cells (with no blood vessel walls). There were undoubted fat cells throughout the tissue. The stroma (intercellular substance) of the mass seemed to be very small in amount.

One border of the section showed very few giant cells and numerous spindle cells, arranged somewhat in whorls. There were limited areas, showing the latter type of cells, which suggested spindle cell sarcoma.

Throughout the entire section there was some evidence of karyokinesis.

Diagnosis (June 9, 1922): Giant cell sarcoma (of epulis type) with beginning malignancy (spindle cell sarcoma).[16]

Fig. 47 (Case 12).—Appearance of arm, three months after operation.

Pathologic Report (Dr. C. Y. White, June 9, 1922).—On tissue (Fig. 52) from second operation, June, 1914, the report was that the tumor consisted of spindle cells not densely packed, fully formed blood vessels, and, enmeshed in the stroma, rather numerous typical giant cells (epulis type). Elsewhere in the section was fully formed fibrous tissue. The whole picture was one of chronic granulation tissue with giant cells.

The arrangement of the spindle cells showed a distinct tendency to formation of whorls. In areas there were small capillaries with normal walls as well as a few larger blood vessels. Throughout the tissue were numbers of large "gigantic cells."

In comparison with the tissue from the specimen removed in 1913, there were not one tenth as many giant cells (epulis type). The spindle cell was the prevailing type of cell, suggesting sarcoma. It was much more like sarcoma

16. Dr. W. H. F. Addison, professor of histology and embryology in the University of Pennsylvania, ventured the opinion that the giant cells in this section (Figs. 50 and 51) resembled megalokaryocytes rather than foreign body giant cells; but as they represent a pathologic and not a normal constituent of bone, he hesitated to differ too positively from experienced pathologists.

than the 1913 tissue. Against sarcoma was the presence in the 1914 sections of well formed blood vessels, both capillary and larger vessels.

Diagnosis: The question as to whether it was to be regarded as chronic granulation tissue with giant cell formation, or chronic granulation tissue with sarcoma formation, was debatable; but the latter diagnosis was favored.[17]

Case 13.—*Myeloid sarcoma of humerus. History.*—J. W., brunette man, aged 37 years, was admitted to the Episcopal Hospital Jan. 19, 1921. The patient was a carpenter. Two years previously he began to experience soreness in the right arm. He rubbed it with liniments and painted it with iodin. Pain was present only when he lifted the arm over his head and when he did heavy work. More than a year previously when he was throwing ashes out of a cellar window, his right arm broke. He went to the University Hospital where a roentgenogram was taken[18] and where he was treated. He was out of work for five weeks, but even after return to work the soreness in the arm and pain during hard work continued. This disability had been growing worse during the last two months.

Ten days previously, while helping his wife pull out a bureau drawer, he noticed a crack in his arm, and was disabled with pain on the slightest motion. He came to the dispensary of this hospital for treatment, where he was under the care of Dr. William Whitaker. Roentgenograms made by Dr. R. S. Bromer, Jan. 12, 1921, showed "a condition which involves the upper third of the humerus, resembling very much a chronic cystic osteitis, with a pathologic fracture at the junction of the middle and upper thirds of the shaft" (Fig. 53).

Examination.—The patient was admitted to the ward, one week later. He was a stout, healthy looking individual, appearing older than his given age. The right arm presented slight palpable bony thickening in the upper third of the humerus, no redness; only slight tenderness on palpation; preternatural mobility and crepitus at the junction of the upper and middle thirds, and no apparent deformity.

Diagnosis: This was cystic osteitis, probably benign; possibly sarcomatous, since at this patient's age purely benign cysts are unusual.

Operation.—Jan. 21, 1921, an incision was made from midway between the acromion and coracoid, downward along the lateral border of the biceps to the

17. These slides were also examined by the following pathologists, whose opinions are appended: Dr. Allen J. Smith, Philadelphia: giant cell sarcoma, locally malignant; Dr. John Speese, Philadelphia: giant cell tumor, more malignant than sections in 1913; Dr. Herbert Fox, Philadelphia: chronic inflammatory tissue; diagnosis of tumor cannot be made from this section but there are areas suggesting giant cell tumor; Dr. James Ewing, New York: giant cell tumor; Dr. J. C. Bloodgood, Baltimore: giant cell tumor; Dr. E. A. Codman, Boston: giant cell tumor, necrotic; Dr. J. H. Wright, Boston: giant cell tumor; Dr. S. B. Wolbach, Boston: giant cell tumor; Dr. F. B. Mallory, Boston: chronic inflammation.

18. Roentgenograms made at the University Hospital in October and December, 1919, shown the author in 1921 by Dr. Pancoast, revealed fracture of the surgical neck of the right humerus. No bone disease was noted in 1919, but reexamination in 1921, with knowledge of the patient's later condition, enabled one to appreciate a slight trabeculation in the region above the surgical neck, and in the second roentgenogram also a slightly greater atrophy and one which extends farther down the shaft than would be expected to follow disuse alone.

lower third of the arm. The thin cortex over the cyst was easily perforated by blunt instruments. Section of the cyst wall and a specimen of the contents of the cyst were sent to the laboratory. The cyst wall was only from 3 to 4 mm. thick. It was fibrous only, with no trace of bone, where the cyst was entered. The contents resembled currant jelly, with small islets of yellowish

Fig. 48 (Case 12).—Fracture of transplant through the most distal screw hole, nearly eight years after last operation.

white fibrous tissue scattered throughout. Such very free (almost alarming) hemorrhage was encountered from the interior of the cyst that preparations were made to apply an Esmarch bandage around the shoulder; but, by rapidly evacuating all the soft tissue from the interior of the cyst and packing it with gauze temporarily, all bleeding stopped permanently.

An aperiosteal transplant (12 by 1.5 by 1.5 cm.) was next cut by circular saw from the subcutaneous surface of the right tibia. In removing this by osteotome, the shaft of the tibia, which seemed unusually brittle, was accidentally fractured obliquely through the crest at the lower end of the transplant, and the transplant itself was fractured transversely near the center. One fragment of this transplant was placed within the bone cavity of the upper

Fig. 49 (Case 12).—Appearance of arm, Feb. 28, 1922, four weeks after removal of plate and screws shown in Figure 48. The dorsal surface of the lower end of the transplant was trimmed down at the same time to improve the contour of the wrist.

fragment of the humerus extending from the interior of the great tuberosity almost to the line of fracture. The second piece of the transplant was used to bridge the fracture of the humerus, being fixed below as an inlay in a slot cut in the anterior surface of the shaft of the humerus below the cyst, and its

upper end passing into the cavity of the upper fragment of the humerus. The section of bone removed from the humerus to admit the tibial inlay was also inserted into the cavity of the cyst (Fig. 54). After closure of the incision in layers, a plaster-of-Paris dressing was applied to the entire upper extremity and chest, with the shoulder abducted about 45 degrees. The time consumed was one hour and forty minutes.

Course.—Feb. 14, 1921, the leg was dressed. The operative wound was healed. Two very small superficial abscesses in the calf of the leg had ruptured beneath the cast. Union in the fracture of the tibia was not very firm.

Fig. 50 (Case 12).—Low power photomicrograph of tissue secured at first operation (1913) showing innumerable "giant cells" (benign) of the epulis type.

March 4, 1921, the leg had been dressed every few days. Today, six weeks after operation, the arm case was removed for the first time. The wound was healed firmly, and there was good union in the humerus. A roentgenogram was taken and a new plaster-of-Paris dressing was applied to the arm and chest. Union was firm also in the fracture of the tibia. The patient was discharged to return to the dispensary for further dressings (roentgenogram, March 4, 1921, Fig. 55).

The gypsum dressing was permanently removed about twelve weeks after operation, and the patient then wore only a gypsum shoulder cap and carried his arm in a sling. Six months after operation he was able to resume light work.

Up to April, 1923, twenty-seven months after operation, there was no evidence of recurrence of the disease. The humerus was firmly united; roentgenograms revealed nearly normal regeneration of the bone, although the shaft of the humerus was still thickened and deformed (Fig. 56); but the man had normal use of the arm, the only disability being from ankylosis of the

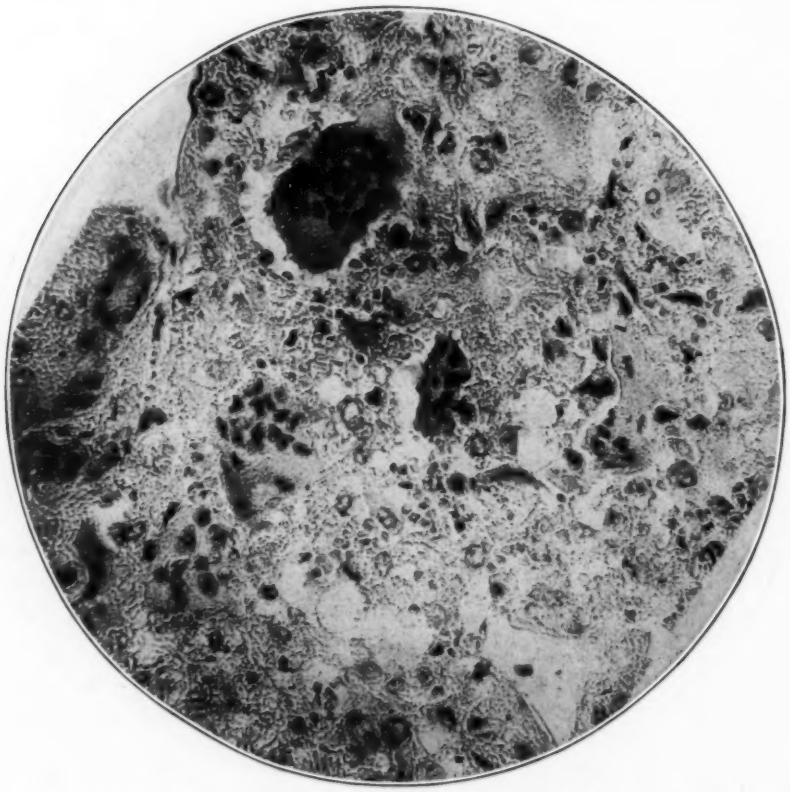

Fig. 51 (Case 12).—High power photomicrograph of tissue secured at first operation in 1913, showing "giant cells" resembling megakaryocytes rather than foreign body giant cells (Prof. W. H. F. Addison, University of Pennsylvania).

shoulder, apparently bony, in abduction about 75 degrees, moderate external rotation and slight flexion.

Pathologic Report (Dr. C. Y. White).—In general, the tumor consisted of spindle cells arranged in irregular whorls, which cells had a large vesicular nucleus. The blood vessels were, in general, fully formed; but occasionally areas were encountered in which blood seemed to be in spaces. Elsewhere there were distinct hemorrhages, and in places this hemorrhage was wide-

spread between the cells. In these areas of hemorrhage there was much blood pigment in granules, some of it inside of phagocytes. In these areas were numerous giant cells of epulis type. These giant cells were less numerous in other areas.

In other sections of the tumor the cells were more crowded. Morphologically, they were of the spindle cell type, representing fibrous tissue cells of granulation tissue. In such areas the giant cells of the epulis type were far more numerous.

In one small area of one section were groups containing from four to five large cells in which the nuclei were centrally located; the cell walls were

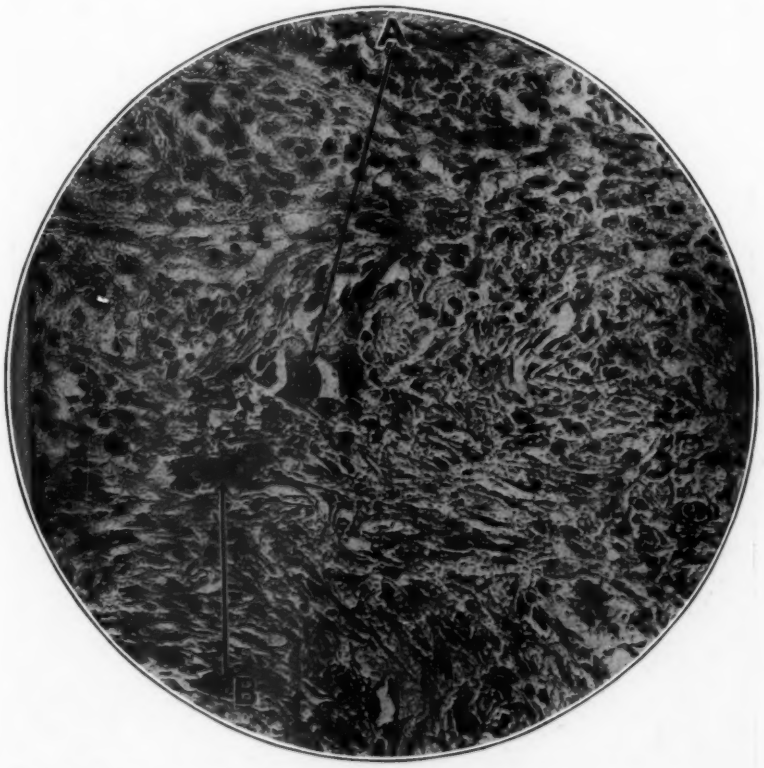

Fig. 52 (Case 12).—High power photomicrograph of tissue from operation in 1914, showing appearances suggesting spindle cell sarcoma, with gigantic cells (true "tumor giant cells"): *A* and *B*, gigantic cells.

irregularly rounded, and the protoplasm did not stain, but showed a very faint reticulation between which was very fine, granular material resembling unstained fat. These cells looked like those described as fatty degenerated cells. Between these groups of fatty cells, the structure of the tumor was similar to that noted elsewhere in the sections; namely, spindle cell.

In sections showing bone, the tumor tissue had encroached on the dense bony trabeculae, infiltrating it in areas.

In some sections an occasional "gigantic" cell was seen.

Diagnosis: This was giant cell sarcoma of bone marrow (giant cell tumor) with areas showing early spindle cell sarcoma (Fig. 57).

These slides were also examined by the following pathologists, whose opinions are appended: Dr. Allen J. Smith, Philadelphia: "myeloid sarcoma, malignant. I have seen two similar cases in which metastases occurred";

Fig. 53 (Case 13).—Cystic osteitis of the humerus.

Dr. John Speese, Philadelphia: giant cell tumor; Dr. Herbert Fox, Philadelphia: spindle cell sarcoma, malignant; Dr. J. H. Wright, Boston: giant cell tumor; Dr. S. B. Wolbach, Boston: giant cell tumor; Dr. F. B. Mallory, Boston: giant cell tumor; Dr. J. C. Bloodgood, Baltimore: giant cell tumor, and Dr. James Ewing, New York: xanthosarcoma.

Dr. Bloodgood's notes made in December, 1921, were:

"It is an unusual picture. It suggests a picture described by Ewing as an endothelial myeloma. The most marked features are groups of palely staining cuboidal and pavement cells of the endothelial type in tubules and irregular cavities, often mixed with blood. Some cavities with blood without tumor cells. This portion is separated by a very cellular stroma, varying in thickness. Cells somewhat of the spindle type. When we look at the tissue surrounding the endothelial area we have a picture of a small spindle and round cell sarcoma. There are a few multinucleated cells. It seems to me we can exclude osteitis fibrosa, the giant cell tumor or inflammatory tissue. In the section there is no bone, no cartilage, no giant cells of the epulis type, nothing to suggest a typical multiple myeloma. The age of the patient, 37, is against osteitis fibrosa."

Fig. 54 (Case 13).—Diagram showing method of insertion of transplants.

Dr. Bloodgood's notes of June 5, 1922, were:

"There are not many giant cells; but the stroma is that of the benign giant cell tumor, or fibrohemangioma of the xanthoma type. . . . I believe this is a benign lesion, central in bone, and should be placed with this group—benign giant cell tumor."

Dr. Ewing's notes were:

"Sections show xanthosarcoma, a cellular variety of the benign giant cell tumor. Some areas contain many large clear cells filled with lipoid granules. Others are very cellular, with compact spindle cells, long and short, nuclei slightly hyperchromatic. There are many blood spaces lined with a few giant cells of the epulis type. This tumor recurs locally. It belongs in the benign giant cell tumor class."

CASE 14.—*Cystic changes in an exostosis springing from the lesser trochanter of the femur in a patient with multiple cartilaginous exostoses. History.*—

Lieut. G. H. M., a brunette man, came under my care in March, 1919, at the Walter Reed General Hospital, Washington, D. C. His previous history was negative. In September, 1917, he was thrown from his horse, and during the three following months he had pain and soreness in the right groin and adductor

Fig. 55 (Case 13).—Cystic osteitis of humerus, six weeks after operation.

region of the right thigh. Gradually, he became aware of the presence of a bony tumor in the latter region, which had steadily increased in size and interfered markedly with his walking. He had a noticeable limp, complained of pain in the right sacro-iliac region, and at each step had to swing his right

lower leg far out from his body to keep the tumor from "interfering." In standing he kept the right hip abducted.

Roentgenographic Examination (Major Selby).—This revealed exostoses over the entire body. The largest, and that which was the cause of the symptoms, was the size of a small orange, and sprang from the region of the lesser trochanter of the right femur (Fig. 58), growing into the adductor region and

Fig. 56 (Case 13).—Humerus, twenty-two months after operation.

butting against the pelvis when attempts were made to adduct the hip. Other exostoses were found in the following situations: left lesser trochanter, lower third of both right and left tibias, middle of left femur and the transverse processes of the fifth lumbar vertebra and on the twelfth ribs. There was hyperostosis of the shaft of the right fibula.

The circumference of the right thigh was 3 cm. greater than that of the left, just below the perineum (59 versus 56 cm.). There was normal motion

in the right hip except adduction, which was lost, and hyperextension and extreme flexion, which were painful. Though there were multiple cartilaginous exostoses, there were none of the deformities associated with the affection known as hereditary deforming chondrodysplasia. The patient's height was 173 cm., and the umbilicus was 99 cm. from the floor.

It seemed to me that in this case the best plan would be to expose first the base of the exostosis at the lesser trochanter. This exposure is not very easy; but I believe then and I believe now that the safest exposure may be obtained by means of Lambotte's incision, which will be described presently. Then, after division of the narrow base of the exostosis, if the tumor proved

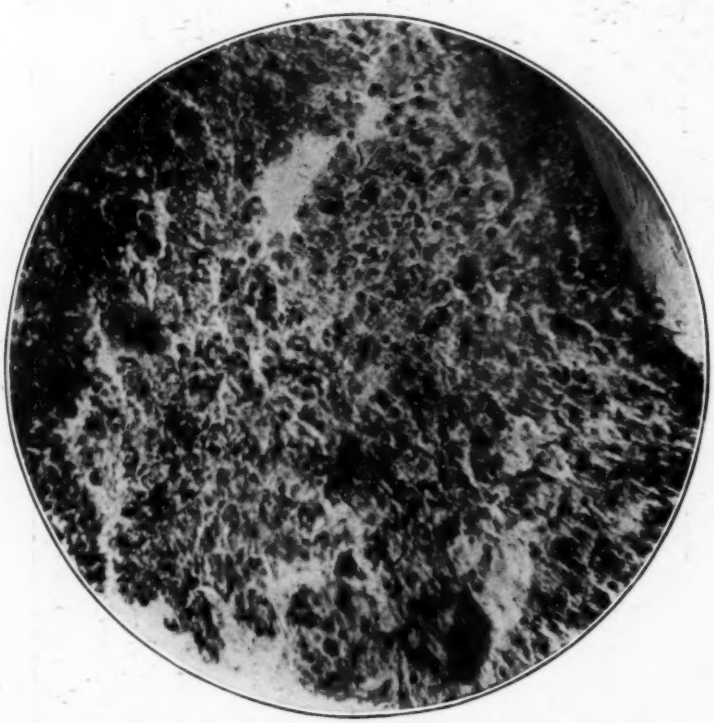

Fig. 57 (Case 13).—High power photomicrograph of tissue secured at operation, showing sarcomatous appearance.

adherent, or if, on account of its size, it could not be removed through the same wound, I determined to make a second incision in the adductor region, similar to that employed by Ludloff in cases of congenital dislocation of the hip, and complete the removal of the tumor by that route. Certainly, by the latter route the lesser trochanter cannot be easily exposed, even in the absence of an overlying tumor.

Operation.—March 13, 1919, at the Walter Reed General Hospital, with the skilful assistance of Capt. John T. O'Ferrall, Lambotte's incision was made from the anterior superior spine of the ilium to the great trochanter, thence downward and forward for a distance of 12 or 15 cm. Then tensor fasciae

femoris and fascia lata were divided in the lines of the skin incision, and the flap drawn to the median side, the retractor protecting the great vessels. The usual approach, on the median side of the long tendon of the rectus femoris, proved so bloody that it was abandoned, and an approach on the lateral side of this muscle was adopted. Before baring the femur, it was necessary to divide the external circumflex vessels. Then the anterior intertrochanteric ridge was exposed, and on the median surface of this the pedicle of the tumor,

Fig. 58 (Case 14).—Large exostosis, with cystic center, springing from right femur near the lesser trochanter.

springing from the lesser trochanter, was identified and divided by chisel. It measured about 3 cm. in its longitudinal diameter and nearly the same transversely. Attempts then were made to enucleate the tumor, but its wall was very easily broken through, and it had to be extracted piecemeal. Its interior was quite friable and soft; its surface was covered with cartilage. The wound was wiped out with hot mercuric chlorid and was closed in layers without drainage. It proved a difficult operation, consuming more than two hours.

Course.—Convalescence was uneventful, and when I returned from active service in the army, at the end of April, 1919, the patient was walking about with support. Under date of June 9, 1922, more than three years after operation, he wrote that the results had been "most encouraging and successful"; and that he had about 75 per cent. of the normal use of his right lower extremity. However, he still complained of some sacro-iliac and lumbar discomfort, perhaps from pressure of the exostosis in that situation.

Laboratory Report.—The specimens removed at operation were examined by Capt. M. W. Lyon, M. C., of the Walter Reed General Hospital, who reported

Fig. 59 (Case 15).—Cystic disease (angioma) of the tarsal scaphoid.

there were no inflammatory cells and no neoplastic cells; there was normal hyaline cartilage, normal osseous material, white fibrous tissue, and a small amount of fatty marrow.

CASE 15.—*Cystic disease (hemangioma) of the tarsal scaphoid. History.*—V. D. A., aged 35, an architect, was seen Sept. 9, 1916, refered to me by Dr. Paul D. Walter of Bethlehem, Pa., and Dr. A. G. Sampson of Philadelphia. The patient had noticed in February or March, 1921, that his right foot and ankle felt as if they had been sprained, but he had no knowledge of any injury. He played tennis without disability during the summer of 1921, but in October of that year the pain in his foot began again. Roentgenograms made at

this time (not seen by me) are said to have shown some bone change, the character of which was not known. His foot troubled him throughout the ensuing winter, and, following an attack of acute tonsillitis in the early summer of 1922, the pain and disability became much worse. Roentgenograms made at this time by Dr. Pfahler (Fig. 59) showed definite cystic changes in the tarsal scaphoid. For the few months previous to visiting me in September, 1922, the disability had been steadily increasing, and for several weeks he had been entirely incapacitated, being barely able to hobble around with a cane. For the previous few days he had suffered acute pain all the time and had been unable to sleep. For many months he had been unable to wear golf trousers, because buckling them around his knee caused such severe pain in the foot.

Examination.—Sept. 9, 1922, the findings were negative except for the condition of the right foot. This showed a prominence on the median border of the scaphoid, so tender that it could scarcely be touched. The astragalus and internal cuneiform bones were not tender, and the rest of the foot appeared normal. Further roentgenograms showed the same condition as in Figure 59. Operation was advised and accepted.

Operation.—Sept. 16, 1922, at the Orthopaedic Hospital, under the diagnosis of "cystic disease" of the tarsal scaphoid, an incision, 10 cm. long, was made on the median side of the tibialis anticus tendon, the incision being curved toward the sole at its distal extremity, so as to turn down a flap of the soft parts. The periosteum of the scaphoid bone was bulging outward on its median surface; the bone beneath felt cystic. The astragalus and internal cuneiform bones were normal. The periosteum of the scaphoid was incised longitudinally on its dorsal surface, exposing, in the interior of the bone, tissue which resembled currant jelly, and throughout which were very friable trabeculae of cancellous bone. From gross examination, it was thought to be a myeloma (giant cell sarcoma). A culture from the interior of the cyst gave no growth, and guinea-pig inoculation was negative for tuberculosis. The interior of the cyst was removed by curet, and specimens of the tissue sent to Dr. E. P. Corson White, pathologist of the Orthopaedic Hospital, and to Dr. C. Y. White at the Episcopal Hospital. These reports are appended. The proximal and median portions of the scaphoid were composed of normal dense cancellous bone. Distally, at the median border, the disease extended as far as the joint with the cuneiform, but the joint itself was not invaded. Thus, the cavity, about 2.5 by 2 cm., in the scaphoid was bounded by apparently normal bone proximally and laterally, and by bulging but intact periosteum medially and distally; the entire median portion of the scaphoid, except where it articulated with the astragalus, was cystic. This cavity was not cauterized in any way, but after thorough curettement was filled with Mosetig-Moorhof's iodoform bone wax. The periosteum was closed with continuous chromic catgut sutures; the deep fascia with interrupted chromic catgut sutures; the superficial fascia with interrupted chromic catgut sutures, and the skin with continuous chromic catgut sutures. Alcohol dressings were applied, and the foot placed in a gypsum splint in a position of slight varus and cavus.

Course.—Oct. 9, 1922, the skin sutures had been absorbed, and the wound was healed except at its middle where it was barely moist. Use of the gypsum splint was continued. The patient, who had been about on crutches since a few days after operation, had had no pain since the operation, even in damp weather; for more than a year before operation his foot had invariably pained him whenever the weather was damp.

Oct. 25, 1922, a roentgenogram showed the wax in the bone cavity; the surrounding bone looked normal. Use of the gypsum splint was discontinued.

Nov. 22, 1922, a little wax had exuded through the center of the incision, requiring change of dressings about once in ten days. The patient had been wearing stout orthopedic shoes for the last week. He still used a crutch on the street.

Dec. 7, 1922, the wound had healed since the last note. The roentgen ray showed considerable bone regeneration and some of the wax still unabsorbed. He walked without a cane.

April, 1923, there were no symptoms.

Pathologic Report.—(Dr. C. Y. White).—The section was composed of fibrous tissue with typical spindle cells, areas showing atrophic bone and numerous various sized spaces filled with blood, and lined with endothelial cells. Also there were large areas showing blood pigment, and a number of giant cells.

Diagnosis: Hemangioma cavernosum of bone (Fig. 60).

Dr. E. P. Corson White reported: The tumor mass invading the bone was made up largely of flat endothelial cells with flat nuclei, which tended to form cystlike cavities often filled with blood. The cavities were backed by connective tissue stroma. Mitotic figures were present but not numerous. In places the cells were collected in more or less concentrated masses, between which fibroblasts had grown. These areas were at times suggestive of malignancy, but probably were not malignant.

Diagnosis: Hemangioma.

Dr. Allen J. Smith reported: The specimen represents ossifying endosteitis; or, if a tumor, telangiectatic osteosarcoma, relatively benign.

DESCRIPTIONS OF ROENTGENOGRAMS BY DR. BROMER

Figure 1 illustrates an area of decreased density, apparently cystic in nature, in the lower third of the tibia along its anterior border, with a definite line (which is evidently proliferating periosteum) outlining the anterior border. The remainder of the shaft shows typical bone changes of syphilis. From the roentgenographic appearance, it is impossible to determine definitely whether this rarefied area has a bony shell as a covering or whether the shadow apparently overlying it is caused by one of the edges of a troughlike erosion caused by the syphilitic process.

Figure 2 presents a cystic condition that is very unusual, involving as it does the diaphyses and the epiphyses of the fibula and the tibia and all bones of the tarsus. There is extreme atrophy of the shafts of the tibia and the fibula, quite frequently seen in congenital syphilis. The involvement of the long bones of all the limbs would tend to indicate that it is a systemic infection. The expansion of the thinned out cortex is abrupt as in giant cell tumors; but in the latter all neighboring bones are never simultaneously involved as are the tarsal bones in this case.

Figure 3, from Case 1, shows the area of osteolysis plainly starting from the medullary cavity and encroaching on the surrounding

cortex, assuming an elliptic shape. There is definite cortical thickening and deposition of new bone. The periosteum shows proliferation; but nowhere is the latter perpendicular to the shaft, the newly formed bone being deposited longitudinally. Knowing nothing of the history of this case, I believe it could be fairly assumed to be an infectious process. The differentiation between syphilis, chronic osteomyelitis and tuberculosis is, however, impossible. The fact that cortical thickening and regeneration are marked would tend to rule out tuberculosis.

Figures 4 and 5 illustrate a cystic condition of bone with trabeculation and the appearance of a multilocular cystic area of rarefaction. However, in the anteroposterior view, there is definite periosteal proliferation laid down longitudinally, which would put it in the class of the infections. There is little tendency to overproduction of bone, and there is the cortical thickening as seen in Case 1. This would narrow the diagnosis to a consideration of tuberculosis alone.

Figure 6 shows a rarefied area, a cystic condition, in the upper extremity of the ulna. It involves both the diaphyseal end and the epiphysis of the olecranon. There is no periosteal reaction. Trabeculae are not clearly defined. There is nothing to indicate tuberculosis, although this was definitely established after operation. It is almost identical to a case of fibrocystic osteomyelitis reported by Barrie,[2] who states that tuberculosis rarely causes fibrocystic changes in bone. In our experience it would seem to be a more frequent cause. He states that these fibrocystic lesions do not increase in size, but that it is impossible to differentiate them from chronic sterile bone abscesses which also do not increase in size.

Bloodgood [8] calls attention to the fact that in a few cases of tuberculous osteomyelitis localized in the shaft of long pipe bones, or in the epiphysis (for example, the lower end of the radius), a central rarefied shadow may be present surrounded by a bony shell with no ossifying periostitis. This case would seem to be an example of such a condition.

In Figure 7 a cystic area, with slight trabeculation, thinning of the cortex and slight expansion in the upper extremity of the humerus, is seen, differing from the picture of chronic cystic osteitis in that the unexpanded portion of the cortex is not involved in the pathologic process. The pathologic fracture can plainly be seen. There is no periosteal reaction, and on this account the infections, syphilis, chronic osteomyelitis and tuberculosis, can be ruled out. The exception to this rule must be borne in mind, however. The cortex does not show the abrupt expansion so often seen in giant cell tumors, and for this reason the latter could be excluded. This narrows the diagnosis to a so-called true bone cyst.

Figures 11, 12, 13, 20 and 21 illustrate cases of multiple fibrocystic osteitis which should not be difficult to diagnose. In these two cases

the characteristic replacement of normal bone detail by irregular strands of trabeculae is seen, enclosing multiple cysts which vary in size and shape. The bony cortex is expanded and thinned. The characteristic deformities, usually bowing of the long bones, with the convexity either inward or outward, are present.

In this connection the observation of Ström [19] should be noted. He cites a case in which the diagnosis of osteitis fibrosa was made only with the greatest difficulty. The lesion was situated in the upper extremity of the humerus, and was characterized by quite extensive deformity of that portion of the bone. The outline of the remainder of the bone was quite normal; but the structure was abnormal, suggesting a cavity formation. The cortical portion in the deformed area was thinned and pressed outward. Close study of the remainder of the shaft revealed the same thinning of the cortex with increase in the marrow cavity, although the outlines of the shaft and the diameter agreed with those of its fellow of the opposite side. He emphasizes the fact that the decisive feature in osteitis fibrosa is not found in the deformed part alone, but in the involvement in the morbid process of the greater portion of the nondeformed part of the bone. This is necessary to rule out the possibility of local neoplastic formation.

The large cyst of the first metatarsal in Case 7 (Fig. 11), involving the entire bone, is merely the picture of a cyst with no distinguishing characteristics. No trabeculae enclosing multiple cysts can be seen. If the patient had not shown the other lesions typical of chronic cystic osteitis, there would be no means whatever of differentiating between it and a so-called true bone cyst.

In Figure 29 the humerus, from its upper diaphyseal extremity downward, for the space of almost half of its length, shows a large cystic area. The cortex is thinned and expanded so that the total diameter is approximately twice that of the normal shaft. Trabeculae are seen. A pathologic fracture is present.

The first condition coming to mind is fibrocystic osteitis, although the so-called true bone cysts show trabeculation. I do not believe a diagnosis of either can be made from the roentgenogram alone. In the unaffected portion of the shaft, the extension of the pathologic process does not show, as it did in the case of Ström.[19] As previously remarked, he considers this finding quite essential in the diagnosis of fibrocystic osteitis.

The pathologist reported "cystic osteitis with giant cells." If he considered it a giant cell sarcoma or myeloma, it certainly serves to

19. Ström, S.: Roentgen-Ray Differential Diagnosis Between Osteitis Fibrosa, Solitary Bone Cyst and Tumors, Acta chir. Scandinavica **53**:21, 1920.

show that the differential diagnosis of these three conditions, true bone cyst, myeloma and fibrocystic osteitis, is impossible by means of the roentgen ray alone.

In Figures 33 to 35 the globular shadow arising from the right side of the fourth and fifth lumbar vertebrae does not show definite characteristics of any new growth. Figure 35 shows slight reticulation. All show a limited outline, as though no invasion was present. A giant cell tumor might be considered.

Figure 39 illustrates Case 11 in which the clinical and roentgen-ray diagnosis was giant cell sarcoma or myeloma. The pathologic report was hardly satisfactory, inasmuch as the type of tumor was not stated. It does not resemble a giant cell growth, inasmuch as there is very slight, if any, expansion of the bony shell, although the dark lines forming trabeculations are present. Granted that expansion is present, it certainly is not the abrupt expansion noticed so frequently in the proved giant cell tumors. If compared with the chondroma carefully studied and reported by Bloodgood,[20] the roentgenographic appearance would seem to be identical.

In Figures 40 and 43 the lesion resembles very closely the typical textbook picture and description of a giant cell sarcoma or a myeloma. It is located at one end of a long bone; viz., the lower end of the radius, one of the bones most frequently affected. The cortex is thinned, the expansion beginning abruptly. The growth is equal in all directions and is reticulated. This, to my mind, is a most typical giant cell sarcoma.

In Figure 53 the lesion shows none of the characteristics of a malignant growth. It rather resembles a chronic cystic osteitis or a myeloma (giant cell sarcoma). The cortex is thinned and expanded. Trabeculae are present. There are shadows of rarefaction in the unexpanded portion of the bone, suggesting chronic cystic osteitis, pointed out by Ström [19] as a distinguishing feature of this condition. The age of the patient would tend, of course, to rule this out. The appearance resembles somewhat, too, a giant cell tumor; but the expansion of the cortex is not quite so abrupt as is often seen in these lesions.

Baetjer [10] gives this description of a typical spindle cell sarcoma of bone: "While spindle cell sarcoma is malignant, it does not seem to be as invasive when seen upon the roentgen-ray plate. It destroys equally in all directions, is generally medullary in origin, does not expand the cortex, but destroys it. The growth, however, does not show the same degree of invasion and may be taken for osteomyelitis. It gives the appearance of being slightly limited in its extension through the medullary canal."

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20. Bloodgood, J. C.: Footnote 8, Page 180, Figure 15.

It can easily be seen that this case presents nothing like the description of Baetjer. Nor can I find in the literature the description of a spindle cell sarcoma presenting a like cystic appearance. The present condition of the patient, more than two years after the operation with no recurrence, would tend to confirm the clinical and roentgen-ray diagnosis rather than the pathologic.

From Figure 58 the diagnosis was comparatively easy, especially since other exostoses were found on examination of the long bones. According to Bloodgood,[8] the exostosis rests upon a foundation of normal bone, while the exosteal growth is composed chiefly of cancellous bone. It may show an outer condensed zone of bone, and there is usually little periosteal bone formation about its base. The roentgen ray reveals little evidence of the cystic condition found at operation.

In Figure 59 the inner half of the tarsal scaphoid shows a cystic formation with trabeculae. There is no invasion of the soft parts. The remainder of the bone seems to be unaffected. No other bones of the tarsus or the foot are involved. This seems to be a case in which a definite roentgenologic diagnosis is impossible. It might be a solitary bone cyst, or possibly a giant cell sarcoma. The latter usually show much more abrupt expansion of the cortex. The lack of invasion would seem to rule out a malignant condition. If the correct diagnosis is cavernous hemangioma or endothelioma, it is the first encountered by us, and serves again to emphasize the impossibility of exact roentgen-ray diagnosis of these lesions.

ARTIFICIAL NERVE BRANCHES FOR INNERVATION OF PARALYZED MUSCLES*

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When a nerve trunk is injured at a level at which important muscular branches are given off, it follows almost invariably that the muscles supplied by these branches are permanently paralyzed, since no path for the conduction of neuraxes from the nerve trunk to the muscle is available. To find a means of forming nerve branches artificially, so that such a path may be furnished for the neurotization of muscles, has been the object of this research. Experimentally, a method has been found whereby a nerve trunk may be made to grow nerve branches at any level in its course and to supply any given muscle which it may formerly have supplied. A muscle normally in the domain of one nerve may be reinnervated by an artificial nerve branch from another nerve without impairing materially the function of the latter.

Artificial nerve branches may be made by using a free autogenous nerve transplant, preferably a small cutaneous nerve, the central end of which is sutured into the nerve trunk and the distal end directly into the muscle to be innervated. By this means, for example, the flexor carpi ulnaris muscle may be neurotized, when its muscular branches have been destroyed, by forming an artificial branch from the ulnar or from the median nerve. Or, if the musculocutaneous nerve is destroyed high up, so that its central end cannot be implanted directly into the biceps, an artificial muscular branch may be made from either the median or the ulnar nerve and implanted into the biceps. The combinations which may be employed are thus rather numerous and are further increased by the fact that more than one artificial branch may be made from a nerve.

The basis of this method lies in principles which are established both experimentally and clinically: First, downgrowth of neuraxes from the central end of a severed nerve trunk can be carried to the distal end by means of a free nerve transplant. Second, if the whole central end of a severed nerve is implanted into a paralyzed muscle, neurotization of the muscle takes place. In the formation of an artificial nerve branch, a small free nerve transplant is sutured to part

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* From the Department of Neurology, Laboratory of Experimental Neurology, Columbia University.

* Read before the combined meeting of the New York Neurological Society and the Neurological Section of the New York Academy of Medicine, Nov. 14, 1922.

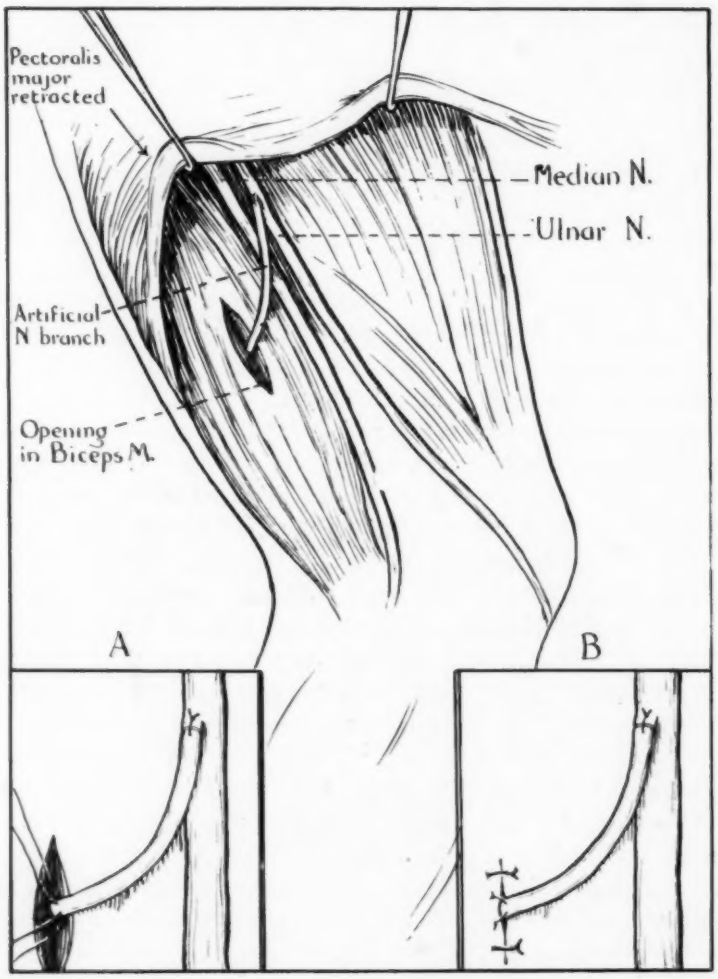

Fig. 1.—Schematic drawing illustrating the method used to form artificial nerve branches. An incision is made in the parent nerve trunk in order to cut sufficient funiculi to cover the cross area of the free nerve segment to be used as a conduction path from nerve to muscle. One through-and-through fine waxed silk suture is passed through the central end to form the central union, and two similar sutures are passed through the epineurium, 2 mm. from the distal end of the nerve segment. The latter sutures are passed into a slit in the muscle as mattress sutures, as shown in *A*. The opening in the muscle is then closed with catgut, and two additional epineural sutures are placed to prevent angulation of the nerve (*B*).

of a nerve trunk and utilized to carry the downgrowing neuraxes directly to the muscle. The free end of the transplant is implanted into the paralyzed muscle just as is the severed end of a whole nerve trunk, and neurotization follows in like manner.

An incision into the nerve trunk is made as if a partial nerve crossing were to be performed, only sufficient funiculi being cut to cover the thickness of the free nerve segment to be used as the artificial branch. A sort of flap is thus formed; but it is raised from the parent trunk only enough to permit accurate end-to-end approximation of the severed funiculi and the free nerve segment.

When neuraxes are cut, we know that in the process of regeneration the central end of a single neuraxis may form a number of neuraxes.

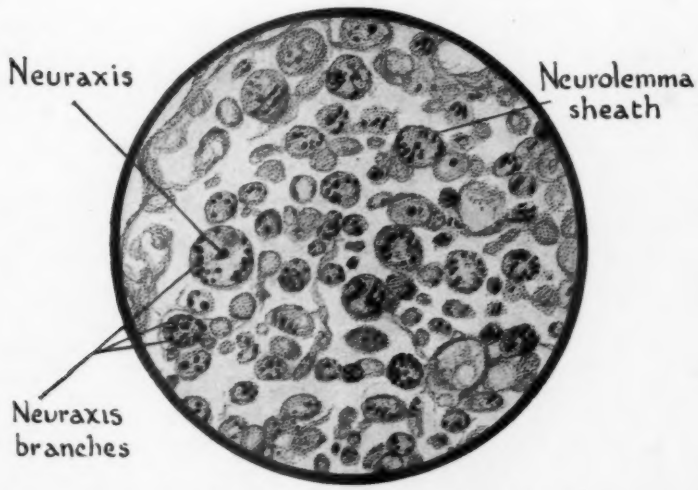

Fig. 2.—Cross-section through a nerve transplant, showing downgrowth of neuraxes and numerous neuraxis branches within a single neurolemma sheath. Ranson pyridin silver stain, paraffin section; × 430.

As many as ten or twenty may be seen in cross-sections within the same neurolemma sheath (Fig. 2). By leaving the nerve flap and line of suture in situ, some of the newly formed regenerating neuraxes which do not grow into the artificial nerve branch may find their way down their old sheaths and regenerate within the distal part of the nerve trunk. While it has not been possible to demonstrate that these neuraxes do find their way into the old sheaths, the possibility of their further downgrowth in them is at least not precluded. Thus, the loss of neuraxes to the nerve trunk may be diminished if the artificial nerve branch is brought to the nerve trunk and if the flap is raised only sufficiently to permit suture.

The distal end of the artificial nerve branch is implanted into the muscle to be neurotized as near as possible to the motor point of the muscle, that is as near as possible to the point where the muscular branch entered the muscle. This point of implantation is to be preferred, since it is the physiologic point of entrance of the nerve. However, it is not essential, since neurotization of the muscle may take place irrespective of this. In selecting the point for implanation, sufficient allowance should be made to prevent tension on the nerve during contraction of the muscle or movements of the extremity.

To implant the distal segment, a slit is made in the muscle in the direction of the muscle fibers and hemostasis obtained by placing a small bit of moist, warm cotton in the opening thus made, and waiting a

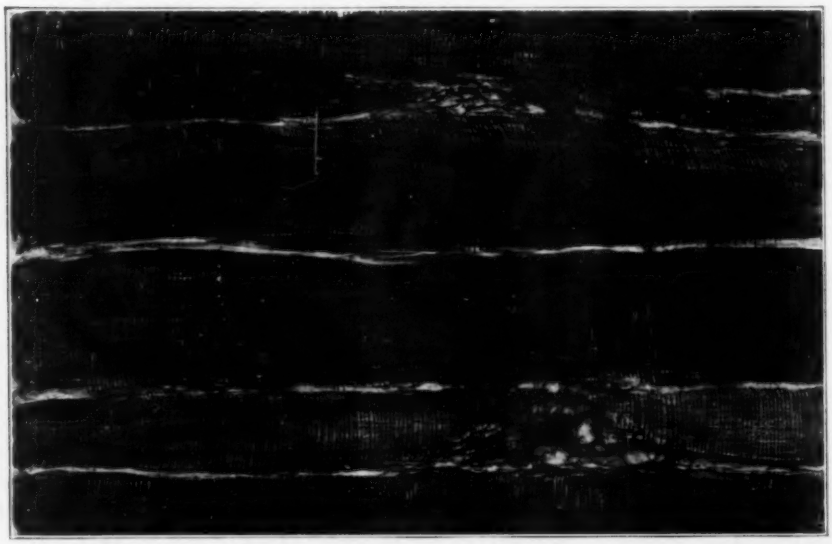

Fig. 3.—Longitudinal section through paralyzed biceps muscle innervated by an artificial nerve branch from the ulnar nerve, 139 days after section of the musculocutaneous nerve. Normal striations and normal size of muscle fasciculi indicate regeneration. Ranson pyridin silver stain, paraffin section; × 430.

moment. Two fine waxed silk sutures are passed through the epineurium of the nerve segment at opposite points, about 2 mm. from the distal end. The nerve end is then buried in the muscle by passing the epineural sutures into the muscle as mattress sutures (Fig. 1). The opening in the muscle is then closed with catgut, and two additional epineural sutures are passed to hold the nerve in place and prevent kinking at the point of implantation.

It is conceivable that, by the time the neuraxes have grown from the nerve trunk through the nerve branch to the muscle, the distal implanted

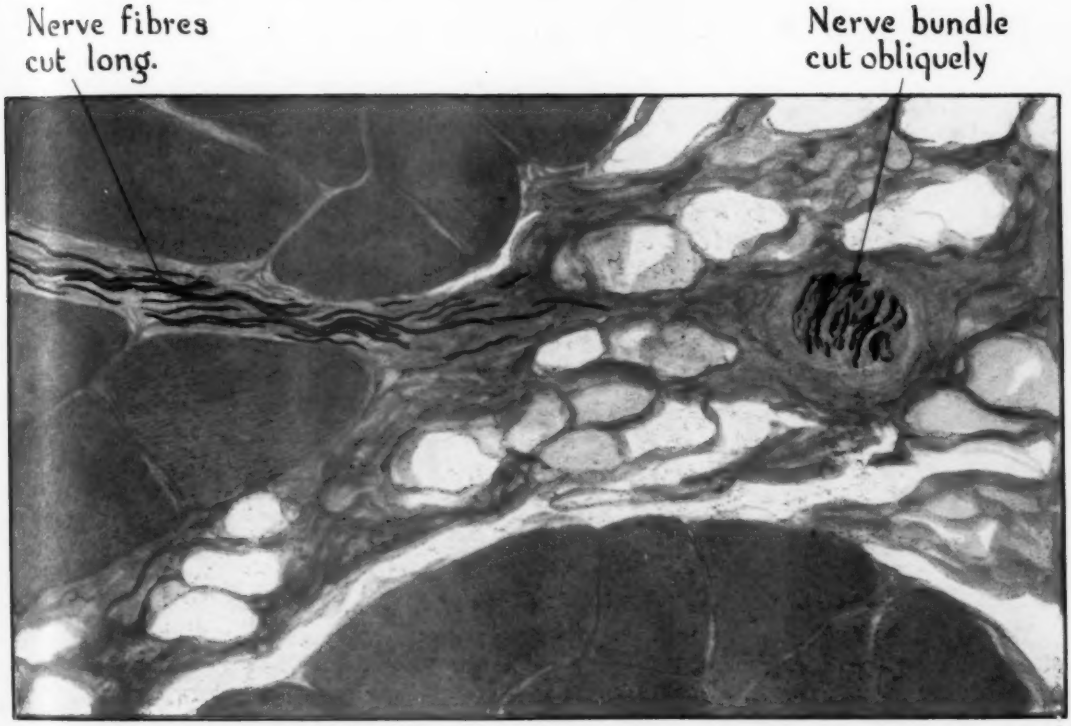

Fig. 4.—Cross-section through paralyzed biceps muscle innervated by an artificial nerve branch from the ulnar nerve, 239 days after formation of an artificial nerve branch. A small nerve bundle is cut obliquely, and nerve fibers are seen passing between the muscle fasciculi. Appearance of muscle, normal. Ranson pyridin silver stain, paraffin section, × 430.

end of the nerve branch may be closed off or blocked by connective tissue, and neurotization prevented. While this is theoretically possible, it has not occurred in our experiments. However, should this contingency arise, the nerve branch may be cut at the point of implantation and reimplanted into the muscle at a higher level. The artificial nerve branch at this time would contain neuraxes. If it is then cut at the point of implantation, neuraxes are ready to grow at once into the muscle at the site of the fresh implantation without waiting for downgrowth from the main nerve trunk to take place. The time interval for ingrowth into the muscle is thus diminished, and neuraxes may stream into the muscle before scar tissue can block them.

In selecting the point on the nerve trunk for suture of the artificial nerve branch, it would perhaps be preferable, on general physiologic grounds, to choose a level near which muscular branches formerly came off for the supply of the muscles to be innervated, though any level may be used and neurotization obtained. If an artificial muscular branch is to be made for a muscle foreign to a given nerve, a point should be selected, so far as possible, some distance from the other branches of the main nerve, so as to allow for rearrangement of the nerve fibers within the nerve trunk. If this precaution is followed, there will be less likelihood of serious interference in the peripheral distribution of the nerve used. Obviously, it is possible that the function of the main trunk may be interfered with, and a definite paralysis may result, even if due regard is had for the funicular anatomy as it is now known. But there are only a few nerves in which the funicular arrangement is definitely determined a great distance from the point at which the muscular branches are given off; for example, the funiculus to the pronator radii teres and flexor carpi radialis which can be traced centrally in the median nerve as far as the union of the outer and inner heads. Yet in most nerves, there is a surplus of nerve fibers, and numerous internal nerve plexuses for rearrangement of the fibers, so that the possibility of a definite paralysis may be diminished though it should always be borne in mind. The possibility of an additional partial paralysis is a point against the method which I wish to stress, and it is also important to point out that with due regard for the neural anatomy and delicacy in technic such paralyses may be reduced to a minimum.

In all, five dogs were used in our experiments. The musculocutaneous nerve was cut immediately after its formation. The central end was then tied with silk to prevent outgrowth of neuraxes, and, as a further precaution, the central end was implanted into the pectoralis major muscle. The site of the implantation was doubly closed by suturing the muscle over the opening in two layers. At the termination of the experiment, the musculocutaneous was stimulated centrally, and in no instance was there any response in the biceps or even in the

pectoralis major, showing that regeneration and ingrowth from the musculocutaneous to the biceps had not taken place, probably owing to the fact that the end of the nerve was tied off.

In some instances in previous experiments, it was found that the biceps muscle may receive a branch from the median nerve, this apparent anomaly is to be expected if the fact is realized that the musculocutaneous in its development at one time was but a branch of the median and that the median may take over a part or the whole of the supply of the musculocutaneous. To prevent any such branch from supplying the biceps, a careful dissection was made along the biceps and the median nerve, and the median nerve was isolated and stimulated throughout to determine the presence of any such branch. In one instance, this branch was found, and it was treated in the same manner as the musculocutaneous, that is, the branch was cut, tied and buried elsewhere.

PROTOCOLS

EXPERIMENT 1 (Nov. 2, 1921).—A medium sized black French poodle was used. The nerves on the inner side of the arm above the elbow were exposed in the usual manner. When the median nerve was traced, there seemed to be two branches, one on either side of the brachial artery. These were traced beneath the artery, where they united at the biceps tendon and passed in the direction taken by the median nerve. The dissection of the biceps was made to make sure that no branches entered from either the median, ulnar or the musculospiral nerve.

The ulnar nerve was isolated in the middle third of the arm, and an adjacent skin branch, 3 cm. long, was freed and cut after a waxed silk suture had been passed at either end. The method of treatment of the musculocutaneous and the technic of the suture were described above.

Comment.—It was extremely difficult to suture the central end of the free nerve segment because the ulnar was extremely small, however, a partial nerve crossing was done satisfactorily. The dog died after twenty-four days. The biceps presented the typical appearance of denervated muscle. No electrical tests were performed because the dog was found dead.

EXPERIMENT 2 (Nov. 3, 1921).—A brown bulldog, with white on its neck, was used. The nerves on the inner side of the arm were exposed in the usual manner. Dissection was made to make sure that no abnormal branches entered the biceps. Treatment of the musculocutaneous and the technic of suture were those previously employed. The point of implantation was 1 cm. below the point of entrance of the musculocutaneous into the biceps. When the musculocutaneous nerve was incised, a larger vessel was cut, and considerable bleeding was encountered. When the sutures were inserted at the point of implantation, a small vein was transfixed. Unfortunately this caused much bleeding at the point of implantation. The dog was killed, March 23, 1922, 139 days after the beginning of the experiment.

A dissection was made of the operative region; the biceps was exposed, and the artificial nerve branch readily seen from its origin on the radial border of the ulnar nerve to the site of implantation. The musculocutaneous was identified centrally and traced to the point where it was embedded in the pectoralis major.

The musculocutaneous nerve was stimulated by a faradic current of various intensities. No response was obtained in the biceps or in any other adjacent muscle. The median nerve was then isolated and stimulated. No contraction was obtained in the biceps, thus ruling out the possibility of the presence of any abnormal branch to the biceps. The ulnar nerve was isolated centrad to the origin of the artificial nerve branch. On stimulation, the biceps contracted forcibly, as well as the ulnar musculature. The nerve was then stimulated below the origin of the artificial nerve branch, and contraction was obtained in the normal peripheral distribution of the ulnar nerve. Stimulation of the artificial branch resulted in a quick response in the biceps. The biceps muscle appeared normal.

Specimens were removed and placed in ammoniated alcohol.

EXPERIMENT 3 (Nov. 5, 1921).—A large white bulldog was used. The technic was the same as that employed in the former experiments throughout. A 3 cm. segment was cut, and the suture of the ulnar nerve was made along the radial border of the latter. The site of implantation into the biceps was on the superficial surface, just below the point at which the musculocutaneous entered it.

Comment.—Both the implantation and the suture were satisfactorily done. There was no bleeding, and good results were expected. The dog was killed, July 20, 1922, 256 days after the beginning of the experiment.

A clean dissection was made of the operative field, exposing the nerves as they arose from the brachial plexus. The musculocutaneous, median and the musculospiral nerves were isolated and stimulated separately. Contraction occurred in the normal distribution of the median and musculospiral; but no contraction occurred in the biceps on stimulation of the musculocutaneous. No response was obtained in either the biceps or the pectoralis major, into which it had been implanted after being tied off.

The ulnar nerve was then stimulated centrad to the origin of the artificial nerve branch. Contraction of the biceps was rapid and complete, causing rapid and quick flexion of the forearm. Stimulation of the branch itself also caused contraction to the biceps. No contraction was noted in the peripheral distribution of the ulnar on stimulation of the artificial nerve branch. Stimulation of the ulnar nerve above and below the artificial nerve branch resulted in contraction in the peripheral distribution.

The biceps muscle appeared normal in size and reddish. There were no fibrillations, and no yellowish color, such as is seen in denervated muscle.

The specimen was removed and placed in ammoniated alcohol.

EXPERIMENT 4 (Nov. 23, 1921).—A large black woolly haired dog was used. The same technic was employed as in the foregoing experiments. The dog was extremely fat, and considerable difficulty was encountered throughout the operation. There was considerable bleeding, and the central union of the nerve segment was not very good.

The dog died after thirty-one days. The muscles presented the typical appearance of denervated muscle. The point of union of the nerve segment with the ulnar and biceps was readily seen, and there was very little scar tissue. The dog died during the night; no electrical stimulations were done.

EXPERIMENT 5 (Nov. 30, 1921).—A large tan and white hound was used. The technic throughout was identical with that employed in previous experiments. There was very little bleeding, and the suture point was satisfactorily determined. The implantation into the biceps was made on the under surface of

the biceps. The whole operation was satisfactory, and good results were expected. The animal was killed, July 27, 1922, 239 days after the beginning of the experiment.

The operative field was exposed in the usual manner, and the nerves isolated as they arose from the brachial plexus. Stimulation of the median, musculocutaneous and musculospiral caused no contraction of the biceps. The musculocutaneous had been implanted into the pectoralis major after it had been tied off, and no contraction took place in the pectoralis major on stimulation of the musculocutaneous.

The artificial nerve branch from the ulnar to the biceps was readily seen. Stimulation of the ulnar nerve centrad to this branch caused contraction of the biceps. Stimulation of the artificial nerve branch caused contraction of the biceps. Contraction in each instance was forcible and caused flexion of the forearm. Stimulation of the ulnar nerve above and below the artificial nerve branch caused contraction in the peripheral distribution of the ulnar nerve. The biceps muscle appeared normal in size and of the normal reddish color. No fibrillations were seen. Specimens were removed and placed in ammoniated alcohol.

CONCLUSIONS

1. When muscular branches are destroyed and nerve suture is impossible, paralyzed muscle may be innervated by the formation of an artificial nerve branch.

2. When a free nerve transplant is sutured to the nerve trunk and the distal end implanted directly into the muscle, the free nerve transplant serves as a conduction path from the nerve trunk to the muscle.

3. An artificial nerve branch may be made for a muscle from a nerve trunk which normally supplies the muscle; or, if this nerve trunk is totally destroyed, a branch may be made from an adjacent nerve. Thus a muscle may be brought under the domain of a nerve which never supplies it normally; for example, the biceps may be supplied by an artificial branch from the musculocutaneous, or, if the musculocutaneous is destroyed, it may be innervated by an artificial branch from the ulnar or median nerve.

4. Evidence that paralyzed muscles may be neurotized by an artificial nerve branch was shown by electric stimulation of the artificial nerve branch, resulting in a rapid and quick contraction of the muscle, by the normal size and color of the muscle, and by the histologic findings, which revealed normal striations in the muscle fibers. The presence of nerve branches and nerve fibers in the muscle thus innervated is conclusive evidence that neurotization has taken place.

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PRESERVATION OF THE FACIAL NERVE IN THE RADICAL TREATMENT OF PAROTID TUMORS *

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Since in our experience facial paralysis has been a common sequela following the removal of malignant tumors of the parotid, we present herewith a description of our technic in removing the malignant parotid gland with the preservation of the facial nerve. We do not advise this radical procedure for simple encapsulated mixed tumors of the parotid; but we are in accord with Sistrunk in urging early diagnosis and early operation in such cases. However, we believe that there is need for a surgical procedure which will permit the complete removal of the parotid gland when these tumors have broken through their capsules into the parotid tissue. It must also be borne in mind that this radical operation should be performed if there is any doubt concerning metastasis from the encapsulated tumor into the parotid gland. Surgical measures should be instituted before the metastasis becomes so extensive as to break down the parotid gland and invade the cervical lymph glands, as it is extremely difficult to preserve the facial nerve when the lines of cleavage have been destroyed. In one case we found that the facial nerve had been invaded by malignant cells, producing a facial paralysis which made preservation of the nerve impossible. This emphasizes the fact that it is better to err on the side of safety by completely removing the parotid gland when in doubt concerning the malignancy of an apparently benign mixed tumor, than to perform an incomplete enucleation, leaving malignant tissue, with a broken capsule.

Volkmann [1] believes that these tumors of the parotid are endotheliomas rising from the endothelium of the blood vessels and lymphatics. Da Costa [2] classifies them as endotheliomas and says that he has removed one such tumor from the tonsil, two from the nasopharynx, one from the superior maxilla and two from the parotid body. He emphasizes the point that it is not possible clinic-

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* From the Section on General and Neurologic Surgery, Mayo Clinic.

* Read before the Western Surgical Association, Minneapolis, Dec. 8, 1922.

1. Volkmann, R.: Ueber endotheliale Geschwülste, zugleich ein Beitrag zu den Speicheldrüsen und Gaumentumoren, Deutsch. Ztschr. f. Chir. **41**:1-180, 1895.

2. Da Costa, J. C.: Modern Surgery, Ed. 8, Philadelphia, W. B. Saunders Company, 1919, p. 427.

ally to distinguish with certainty these endotheliomas from ordinary sarcomas. Fraser [3] asserts that mixed tumors originate from the glandular structure of the salivary glands, and in his article, based on the experimental production of the neoplasm in the submaxillary gland of the dog, he concludes that mixed tumors arise from the ducts of adult glands and that injury causing partial obstruction of these ducts probably plays an important part in the production of the tumors. New [4] believes that, since it is impossible to demonstrate any close connection, either grossly or microscopically, between the salivary gland and the tumor, they probably do not arise from the glandular substance, particularly since the tumor, in its incipiency, is surrounded by a definite capsule and appears to be situated superficially on the surface of the gland. He believes also that if trauma to the ducts of the parotid gland is a causative factor, it should be more common in the submaxillary region than in the parotid, since the submaxillary ducts are more commonly affected by salivary calculi and acute and chronic inflammatory conditions than are the parotid gland and Stenson's duct. Certain pathologists believe that they originate from fetal rests in the parotid which are encapsulated and which gradually grow. Wilson and Willis,[5] after a study of tumors of this type, obtained from operations in the clinic, concluded that they are mesotheliomas. Still others believe them to be carcinomas and basal-cell epitheliomas. The tissue on the interior of the capsule of such tumors varies, a few being composed chiefly of epithelial elements, others have a large amount of fibrous tissue, while in others tissue resembling cartilage seems to predominate. It is not unusual to find all three of these tissues represented in the same tumor. Such tumors are not confined to the parotid alone, but are found in the larynx, pharynx, palate, upper lip, sublingual region, cheek and submaxillary regions. In New's series of sixty-eight cases, fifty of the tumors were of the parotid, one of the larynx, four of the pharynx, three of the palate, three of the upper lip, one of the cheek, one of the sublingual region and five of the submaxillary.

Mixed tumors of the parotid constitute a very small percentage of malignant tumors of the body. Sistrunk [6] found that they occurred in 0.062 per cent., or one in every 1,607 patients who were examined in

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3. Fraser, A.: Mixed Tumors of the Salivary glands: A Study Based on the Experimental Production of Neoplasm in the Submaxillary Gland of the Dog, Surg., Gynec. & Obst. **27**:19-39 (July) 1918.

4. New, G. B.: Mixed Tumors of the Throat, Mouth and Face, J. A. M. A. **75**:732-735 (Sept. 11) 1920.

5. Wilson, L. B., and Willis, B. C.: The So-Called "Mixed" Tumors of the Salivary Glands, Am. J. M. Sc. **143**:656-670, 1912.

6. Sistrunk, W. E.: Mixed Tumors of the Parotid Gland, Minnesota Med. **4**:155-160 (March) 1921.

the Mayo Clinic from 1915 to 1919. The incipient growth is a painless, small round, or irregular, firm tumor in the parotid region, and is usually discovered accidentally. If the tumor is left alone, the extent of its growth varies. In some instances, it is rather rapid, in others, the tumor does not seem to change in size for a number of years. But, sooner or later, it enlarges to a noticeable size, and the patient is then likely to consult a physician. Such tumors may exist for years (forty in one case) or only for a few weeks; but when they take on new growth the problem becomes serious, for sooner or later the capsule will break and the parotid gland be invaded. As long as the tumor remains encapsulated, it is not highly malignant; but when its capsule is ruptured through growth, or through an incomplete operation in which the entire tumor is not removed, invasion of the surrounding tissue soon takes place, and the growth becomes highly malignant. Sistrunk emphasizes the importance of early diagnosis and early operation in cases of incipient tumors. He says that complete removal of the growth at this stage will be followed by permanent cure in practically every instance. We concur fully in this, but we wish to emphasize the importance of complete removal of the parotid gland if there is any possibility of not being able to make a clean enucleation of an apparently benign tumor, or if there is any evidence of an extension of the tumor through the capsule into the parotid gland, rather than waiting for recurrence. Radium is, no doubt, of distinct value in treatment; but its effects do not compare with surgical measures in the treatment of these tumors, since they are not highly malignant at the outset.

OPERATION

Sistrunk calls attention to the importance of exposing the facial nerve before removing an encapsulated tumor of the parotid gland. He locates the facial nerve by first isolating the inframandibular branch of this nerve as it runs along the angle of the jaw and over the facial vessels. He then follows the inframandibular branch upward through the substance of the parotid gland to the point where the facial nerve divides into the temporal and cervical branches. The lower lobe of the parotid is then elevated, and the finger introduced between the parotid and the nerve. He further asserts that the surgeon may feel sure that enucleation is being carried on in a plane lying above the nerve, and, although a temporary paralysis generally occurs from trauma to the nerve, it usually disappears within a year. We have found that it is possible to carry the dissection of the temporal and cervical portion of the seventh nerve through the parotid and on the muscles of the face by elevating the remaining portion of the parotid

gland and dissecting the nerve away from the gland. This is possible by making an additional incision downward from the zygoma, just in front of the ear, but behind the parotid gland. The incision intersects the curved incision that has been made to identify the inframandibular branch of the seventh nerve. The latter incision extends from 3 cm. upward and posterior to the lower tip of the mastoid process and runs 2 cm. below the lower border of the mandible, ending 4 cm. anterior to the angle of the jaw. After this incision has been made

Fig. 1.—Incision for the removal of the parotid gland.

through the skin, the inframandibular branch of the seventh nerve should first be exposed and then dissected upward until the temporal and cervical divisions of the facial nerve are exposed. The dissection should then be carried from above downward in the line of the incision from the zygoma, and should be deep enough to expose the mandible, the temporal vessel and the posterior border of the masseter muscle. At first this dissection should be made through a narrow field to

avoid injury to the temporal branches of the facial nerve, until one is able to connect this surgical field with that exposed by the cervical incision described above. The next step is a dissection of the cervical and temporal divisions of the seventh nerve through the parotid. It is surprising to learn that the facial nerve penetrates the lower lobe of the parotid only for a distance of approximately 2 cm., after which the nerve lies underneath the parotid on the muscles of the face. From this point, the parotid together with the skin may be elevated,

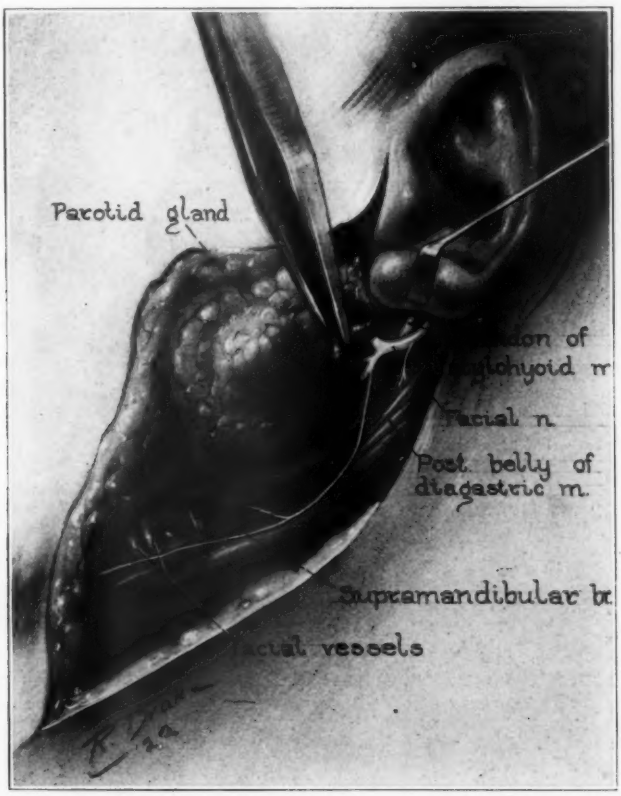

Fig. 2.—An elevation of the inferior lobe of the parotid exposing the facial nerve with it; main divisions and the supramandibular branch.

leaving the peripheral branches as they lie undisturbed on the facial muscles. The dissection is then carried forward sufficiently to elevate all of the parotid, and, when this is completed, Stenson's duct is ligated and divided and the parotid gland removed from the skin. If a skin area is involved, the involved area may be removed with the malignant mass. The deep lobe of the parotid which lies posterior and mesiad to the ramus of the mandible is removed by gently elevating

the facial nerve outward and dissecting out the parotid gland which lies mesiad to it. The exposure obtained by the foregoing procedure facilitates complete removal of all parotid tissue with any involved skin that may be present, without severing or injuring the facial nerves. The dissection must be done gently, and traction should not be exerted on the nerve, nor should it be handled with heavy forceps. It can be elevated gently and the nerve trunks may be lifted with dural forceps,

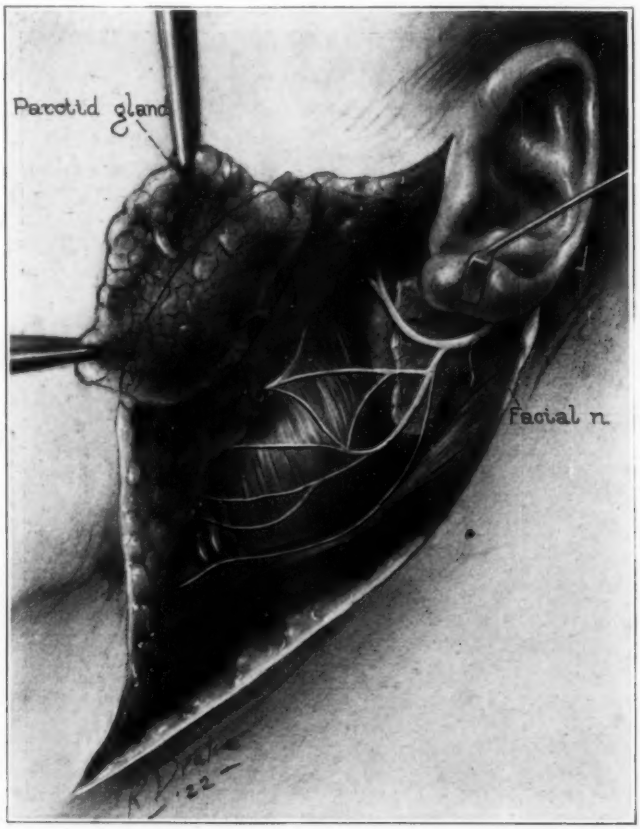

Fig. 3.—The facial nerve passing through only a small portion of the parotid after which it lies on the muscles underneath the parotid gland.

care being taken to apply the forceps to the perineurium. Occasionally, slight difficulty is encountered in handling the temporal artery, as it lies just in front of the ear, under the border of the parotid gland, and passes downward and inward underneath the temporal and cervical divisions of the seventh nerve. Caution should be exercised to keep the incision made from the zygoma downward close to the cartilage of the ear, and to carry it just in front of the tragus down to the

lobule, curving it backward and upward to meet the incision which extends from the mastoid below the mandible, thus avoiding any necrosis which might take place from a too pointed flap of skin just below the ear. A better exposure is obtained if a few of the fibers of the sternomastoid are divided on the anterior border at the mastoid attachment. Gentle traction on the lobule of the ear in an upward and backward direction facilitates exposure of the facial nerve. The

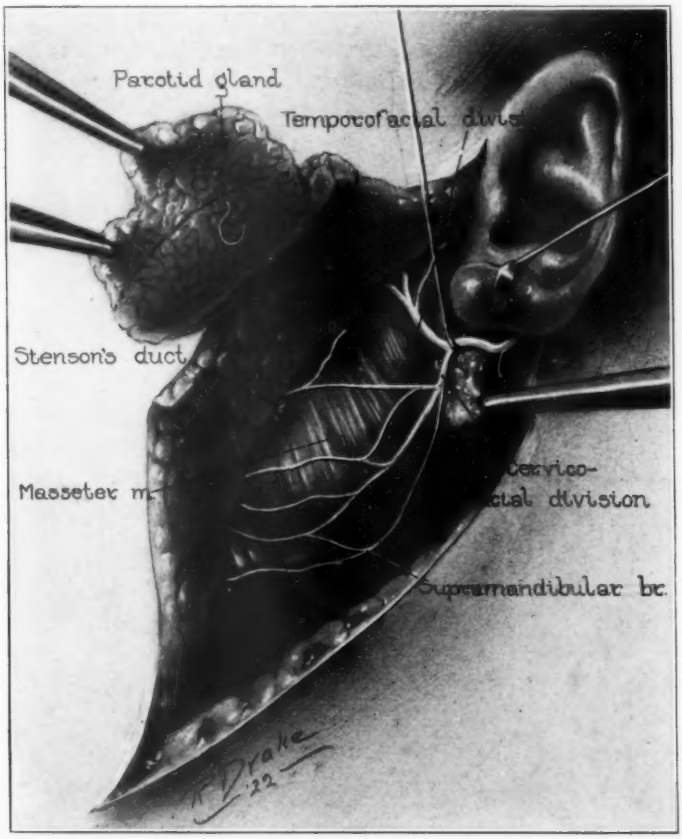

Fig. 4—Preservation of the facial nerve during the removal of the posterior lobe of the parotid.

wound is closed with a subareolar stitch of catgut and with a dermal stitch of horsehair. Drainage can be employed at the lower angle of the cervical wound if necessary. Every precaution should be taken to avoid sepsis or unnecessary hemorrhage by carefully preparing the field with iodin and plugging the external auditory canal with sterile absorbent cotton after the external ear has been prepared with iodin. The bleeding of the skin and parotid can be diminished by injecting

a weak solution of epinephrin along the line of the incision. Postoperative care is the same as that advised for patients on whom dissections of the neck and face have been performed (Figs. 1, 2, 3 and 4).

SUMMARY

Early diagnosis and early and complete enucleation are advised for encapsulated tumors of the parotid gland; but, if metastasis to the gland has occurred, radical removal should be carried out with preservation of the facial nerve. The facial nerve should be sacrificed only if metastasis and necrosis have become so extensive that it is impossible to demonstrate the lines of cleavage.

THE TREATMENT OF BRAIN ABSCESS BY THE INDUCTION OF PROTECTIVE ADHESIONS BETWEEN THE BRAIN CORTEX AND THE DURA BEFORE THE ESTABLISHMENT OF DRAINAGE

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Although medical literature is replete with reports of isolated cases of brain abscess with recovery, the percentage of recovery would doubtless be distressingly small were the full reports of all such cases made known. In reviewing the literature, one is impressed with the lack of uniformity of technic in the operative treatment of brain abscess, as well as with the lack of emphasis placed on certain fundamental principles, the observation of which is essential in order to obtain a reasonable degree of success.

In 1920, Lemaitre [1] called attention to the importance of walling off the subarachnoid space in the treatment of brain abscess. Since the publication of his article, I have adopted the method described below, which has been used with a degree of success sufficient to warrant its publication.

There are two complications in the treatment of brain abscess which are responsible for most of the poor results; namely, meningitis and brain fungus. Meningitis results, as a rule, from the contamination of the subdural and subarachnoid spaces at the time of, and subsequent to, the establishment of drainage of the abscess; and brain fungus occurs when a too extensive opening has been made in the skull and dura.

The contamination of the subdural and subarachnoid spaces can be largely prevented if adhesions between the brain cortex and dura are induced before permitting the free evacuation of the abscess cavity. This can be readily accomplished provided the technic described below is rigidly adhered to. After these protective adhesions have been formed, free drainage can be accomplished without fear of meningeal contamination, a principle which has been recognized for many years in the treatment of abscess of the liver, lungs, etc.

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1. Lemaitre, F.: Walling Off the Subarachnoid Spaces: Its Application to the Treatment of Cerebral and Cerebellar Abscesses of Otitic Origin and Also in a General Way, to Surgery of the Brain, Ann. Otol., Rhinol. & Laryngol. **29**:1 (March) 1920.

Brain fungus can hardly occur unless an opening is made in the skull and dura, of a size sufficient to allow the brain to protrude. If the necessary opening is made just large enough to permit the entrance of the exploring needle and for the small drains to follow, the brain meets a natural barrier instead of an opening and cannot, therefore, escape.

TECHNIC OF OPERATION

Before selecting the point of attack, every reasonable method for accurate localization should be used. An entrance through contaminated areas (frontal sinus or mastoid) should be avoided for two reasons: First, the diagnosis of abscess may prove wrong; second, the abscess, if present, may not be reached with the exploring needle. By taking this precaution, the danger of producing an infection of the cranial contents will be obviated.

The incision should be just long enough to permit the entrance of a perforator and small burr. It should be made as nearly as possible over the suspected site of the abscess. After a small opening has been made through the skull, the dura is incised, the incision being just long enough to assure the operator that a cortical vessel will not be injured by the exploring needle. An ordinary straight ventricular puncture needle, with centimeter markings, is then inserted in the direction of the suspected abscess. As soon as the abscess is reached and a few drops of pus have escaped into a sterile test tube, the needle is withdrawn slightly until the pus ceases to appear. The exact depth and direction of the needle are then calculated.

The prevention of the free escape of pus at the time of operation cannot be too strongly emphasized. In the first place, a contamination of the wound is to be avoided, a condition which can hardly be prevented if the pus is allowed to escape freely through and around the needle. In the second place, the thorough evacuation of the contents of the abscess at this time may relieve the increased intracranial pressure to such an extent as to allow the brain cortex to lose its firm contact with the dura at the point of incision and to permit infection of the neighboring meninges. Such a spread of the infection can be prevented if the cortex becomes firmly adherent to the dura around the dural opening before free drainage is established.

After the depth and direction of the abscess have been calculated, the needle is entirely withdrawn, and a small filiform drain of folded rubber tissue is inserted down the track of the needle in the direction of, but not into, the abscess. The wound is then covered with a properly fitted dressing, which is not changed for two or three days. During this time, the rubber tissue wick, acting as a foreign body, stimulates the formation of adhesions between the brain cortex and the dura, forming a well-defined ring around the drain. At the first dressing, the wick

may be inserted into the abscess. This usually causes very little drainage unless the pus is under great tension. Two days later, the second dressing may be performed, and a second small rubber tissue wick inserted down the track along the side of the first. The dressings may then be changed daily, for by this time the protective adhesions are widespread and firm enough to permit the gradual enlargement of the drainage track by means of additional small wicks of rubber tissue. Within a week or ten days, it is safe to withdraw these wicks and to substitute a drainage tube (preferably the eye portion of a No. 10 English rubber catheter) down the well-formed drainage track into the abscess. Usually, free drainage does not take place until tube drainage is established. The dressings from this time may be performed as frequently as desired, the tube being removed and cleansed if suspected of being clogged with exudates. No undue haste in permanently withdrawing the drain should be practiced. As long as there is any drainage whatsoever, the tube should be kept in situ and only removed permanently when all evidences of the infection have subsided. It is a safe practice to leave the tube inserted for a week or ten days after active drainage has ceased, and gradually withdraw it by daily shortening. It may be two or three months before the drainage tube may be dispensed with, although the average case does not require such a long time.

REPORT OF ILLUSTRATIVE CASE

The following case of a small series has been selected to illustrate the value of the method of treatment described above.

HISTORY

M. D., aged 17 years, referred by Dr. C. A. Thigpen of Montgomery, Alabama, was admitted to the Piedmont Sanatorium, July 28, 1922. June 27, the patient first consulted Dr. Thigpen, whose letter concerning her condition is as follows: "On the evening of June 27, she was brought to me in a prostrate condition. What attracted attention at first inspection was a closed right eye with much edema and exophthalmos, the typical picture in children of acute fronto-ethmoiditis. Temperature was 103 F., pulse 120. I operated without delay, making a brow incision. The right frontal sinus was small but filled with thick creamy pus and some granulations. At the same time, I removed the anterior end of the right middle turbinate, broke down the anterior ethmoid cells and established free communication with the frontal cavity. Relief was prompt, the temperature and pulse gradually returning to normal; headaches ceased, and, on the ninth day, she was able to leave the hospital. About a week later, she began to have increased temperature and a return of the headaches, necessitating the reopening of the frontal wound and the curetting away of the granulations. The wound has been kept open ever since. The improvement was again prompt and continued so until three days ago when she went into a state of collapse, with slow pulse, cool extremities, etc. At this time, the optic disks were beginning to show evidence of increased intracranial pressure. I, therefore, have made a diagnosis of brain abscess." She had suffered from fairly constant

headaches, excepting for a few days following the operations, since the onset of the present illness. They had been located principally in the right frontoparietal region. Vomiting, projectile in character, had occurred several times during the preceding week. Double vision, lasting two days only, had occurred five days previously, but was not present at the time of examination. Three or four attacks of dizziness, momentary in duration, had occurred during the previous two weeks, causing her to grasp an object to keep from falling, and had been relieved by lying down. The family history was unimportant, except for arrested "lung trouble" in the mother. The patient had had influenza five years previously, the usual diseases of childhood, and tonsillitis, four years previously.

NEUROLOGIC EXAMINATION

Head.—There was an operation scar, with a small discharging sinus (mucopurulent), on the right side of the bridge of the nose; slight, though definite, dilatation of the veins over the right frontal region; slight edema of the upper eyelids, more marked on the right than on the left. The veins of the upper right eyelid were quite prominent. Both eyes showed a mild degree of exophthalmos, more marked on the right than on the left.

Cranial Nerves.—The retinal veins were greatly engorged. The optic disk was completely obliterated, presenting the picture of a recent, though definite choking. The same conditions obtained on the left as on the right, though to a less marked degree. There was a moderate degree of photophobia on both the right and left sides. No changes were found in the visual fields. The pupils were equal, and reacted to light and in accommodation. All external eye movements were well performed, except when the patient looked to the extreme left. There was no nystagmus. Voluntary and emotional movements of the face were slightly sluggish on the left, as compared with the right. All other cranial nerves functioned normally.

Cerebrum.—There was a fine tremor of the outstretched fingers of the right hand, and no disturbance of memory, except in regard to the recent operation, concerning which the patient had no clear remembrance. There was no frank disorientation. The patient was right-handed. There was no aphasia, no uncinate gyrus symptoms, and no hemianopsia. No convulsions, either general or localized, had occurred. There was no paralysis nor weakness of the various groups of muscles of the extremities on the right or left. Slight weakness of the left facial muscles was observed. Ankle clonus was not obtained either on the right or left. No sensory disturbances were observed on the right or left. There was no astereognosis or disturbance of joint sense.

Cerebellum.—No asynergia was demonstrated on the right or left. There was no nystagmus.

Reflexes.—The abdominal and epigastric reflexes were active and equal on the two sides. The arm reflexes and knee jerks were hyperactive on the right and highly exaggerated on the left. The Achilles jerk was hyperactive on the right and highly exaggerated on the left. Babinski, Gordon and Oppenheim reflexes were absent on the right and left. Kernig's sign was absent on the right and left.

Sphincters.—There was no disturbance of the sphincters.

GENERAL EXAMINATION

General Condition.—At the time of examination, the pulse was 64; the blood pressure: systolic, 98; diastolic, 50, and pulse pressure, 48.

Urinalysis.—The reaction was acid; specific gravity, 1.014. Albumin, sugar and indican were absent. Numerous epithelial cells, a fair number of pus cells and an occasional red blood cell were demonstrated in a noncatheterized specimen.

Blood.—Examination revealed: hemoglobin, 80 per cent.; erythrocytes, 4,250,000; leukocytes, 8,900. Differential count: polymorphonuclears, 69 per cent.; small mononuclears, 30 per cent.; transitionals, 1 per cent.

COMMENT

The development of symptoms and the finding of increased intracranial pressure subsequent to a verified suppurative fronto-ethmoiditis were strongly suggestive of brain abscess. The headaches on the right, the greater choking

Fig. 1.—Lateral view of skull with grooved director passed through the opening in the skull, down the drainage track into the abscess.

of the right optic disk than of the left, the fine tremor of the outstretched fingers of the right hand, the exaggerated deep reflexes on the left, and the slight weakness of the left facial muscles suggested that the most likely site of the abscess was the right frontal lobe.

OPERATION

Under local anesthesia, induced by 1 per cent. procain-epinephrin solution infiltration, an incision, 2 cm. long, was made along the hair line in the right midfrontal region. The opening into the skull was accomplished with a perforator and small burr. An incision, 0.5 cm. long, was made in the dura.

Through this small opening, a straight ventricular puncture needle was passed through the cortex of the brain, downward, slightly forward and mediad. At a depth of 3 cm., a slight resistance was encountered, which was interpreted as being the capsule of the abscess, or possibly the dura overlying an epidural abscess. The needle was gently pushed through the resistance, and pus escaped under great pressure. In spite of the immediate withdrawal of the needle, 10 c.c.

Fig. 2.—Postero-anterior view of skull, with the grooved director passed through the opening in the skull, down the drainage track into the abscess.

of pus had escaped into a sterile test tube before the flow was stopped by withdrawing the needle. A small filiform wick of folded rubber tissue was then inserted down the track of the needle, to the capsule but not into the abscess. The edges of the scalp wound were whipped over with continuous catgut in order to control hemorrhage, and a plain gauze dressing was applied.

DIAGNOSIS

There was a large abscess at the base of the right frontal lobe, 3 cm. below the cortex, at the site of the incision. The pus was thin and greenish. Culture revealed *Staphylococcus aureus*. The brain was under greatly increased tension.

COURSE

July 29, 1922: The dressing was changed, but the drain was not disturbed. Practically no drainage had occurred, and the general condition was good.

July 31: The dressing was changed, and a drain was inserted for 0.5 cm., causing an immediate escape of a moderate amount of pus. The temperature and pulse were normal. There was no evidence of meningitis and no change in the disks. The headaches were not so severe, but they had not been completely relieved.

August 2: The dressing was changed. Drainage had saturated several thicknesses of gauze, but the flow was not free. The drain was manipulated slightly but was not changed. The condition of the patient was excellent.

August 4: The dressing was changed. A slightly larger rubber tissue wick was substituted for the one inserted at the time of operation. Drainage was satisfactory, though not very free.

August 5 to 9: Daily dressings were performed. Drainage was fair but not abundant.

August 10: The drain was removed, and a grooved director was inserted through the drainage track into the abscess. As soon as this was done, fully 30 c.c. of pus escaped. A roentgenographic examination was made with the director inserted into the abscess (Figs. 1 and 2). The director was withdrawn, and a very small rubber catheter drain was inserted into the abscess.

August 12: There was no drainage. The tube was removed, and a No. 10 English rubber catheter drain was inserted, causing immediate free drainage.

August 13 to 24: Daily dressings were performed. Abundant drainage had occurred, which had gradually become less and less. The patient had no symptoms.

August 25 to September 17: The dressings were changed every three or four days. At each dressing, the tube was shortened slightly. Practically no drainage had occurred. The tube was removed completely, September 17.

September 24: The patient was discharged from the hospital. The wound was healed. All symptoms and neurologic findings were absent. Her condition was excellent.

October 4: A letter from Dr. Thigpen was received to the effect that the patient was in excellent condition and apparently cured.

COMMENT

This case is reported fully because it is a typical illustration of what should be expected in most cases of single abscess of the brain, when the abscess can be located with the exploring needle. Large openings into the skull and dura with the immediate establishment of free drainage, I believe, are responsible for the majority of poor results in this type of lesion, as the development of meningitis and brain fungus are to be expected under such conditions. If preformed adhesions between the dura and cortex are present, immediate drainage might be effected with safety. Such a condition, however, is rarely encountered, except in

cases of abscess following injury caused by some foreign body. If protective adhesions are not already present, they should be induced before free drainage is established.

Decompression in cases of brain abscess is particularly to be condemned. As soon as the increased intracranial pressure under such circumstances is relieved by decompression, there is a tendency for the abscess to be dislocated in the direction of the decompression, thereby promoting further destruction of brain tissue, which should not occur if the abscess is drained through a very small opening in the skull.

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PRIMARY LYMPHOSARCOMA OF THE PROSTATE

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In the pathologic laboratories of Bellevue Hospital, I have recently studied a primary tumor of the prostate with the characteristic structure of a lymphosarcoma. With the exception of an almost identical growth recorded by Coupland,[1] I have been unable to find an acceptable description of another primary lymphosarcoma of the prostate in the literature. However, these two cases are so clearly defined that they remove all doubt as to the possibility of lymphocytic tumors' arising in the prostate. It remains to be shown by future investigation just how common such tumors are. I suspect that they are not so rare as the literature would indicate, and, indeed, I believe that lymphosarcoma should always be considered a possibility in patients under 35 years of age with prostatic tumors. The recognition of this type of growth in the prostate is peculiarly important in view of the favorable results that have been recorded by the roentgen-ray and radium treatment of lymphosarcomas in other parts of the body.

Coupland's case was that of a man, 29 years of age, who complained of pain at the point of the penis and an almost constant desire to micturate, passing only a few drops of urine at each time. The urine contained a large quantity of pus. Rectal examination was painful and revealed almost uniform enlargement of the prostate, which was firm but not very tender.

At necropsy, it was found that the bladder resembled a small cone with a broad base. The cavity was not much larger than a walnut and the bladder walls were thickened to the extent of about one-half inch (1.27 cm.). The prostate was the seat of a new growth which was ovoid, and measured, internally, 8 inches (20.3 cm.) in circumference and 3½ inches (8.9 cm.) in length. The growth was fleshy in consistency and surrounded the neck of the bladder and the prostatic urethra. The pancreas was fibrotic, and, at a point corresponding to the junction of the head of the organ with the body, the glandular substance was replaced by an ill-defined, translucent, pale yellow nodule, about the size of a filbert and of fleshy consistency. The right suprarenal was enlarged to about twice the normal size and was uniformly infiltrated by grayish yellow, semitranslucent, fleshy tumor material.

1. Coupland: Tr. London Pathological Society, 1877, p. 179.

The histologic description of the tumor in the prostate, suprarenal and pancreas is that of a typical lymphosarcoma and was so accepted by the Morbid Growths Committee of the London Pathological Society. The illustration which accompanies Coupland's paper can scarcely leave any doubt as to the correctness of this interpretation.

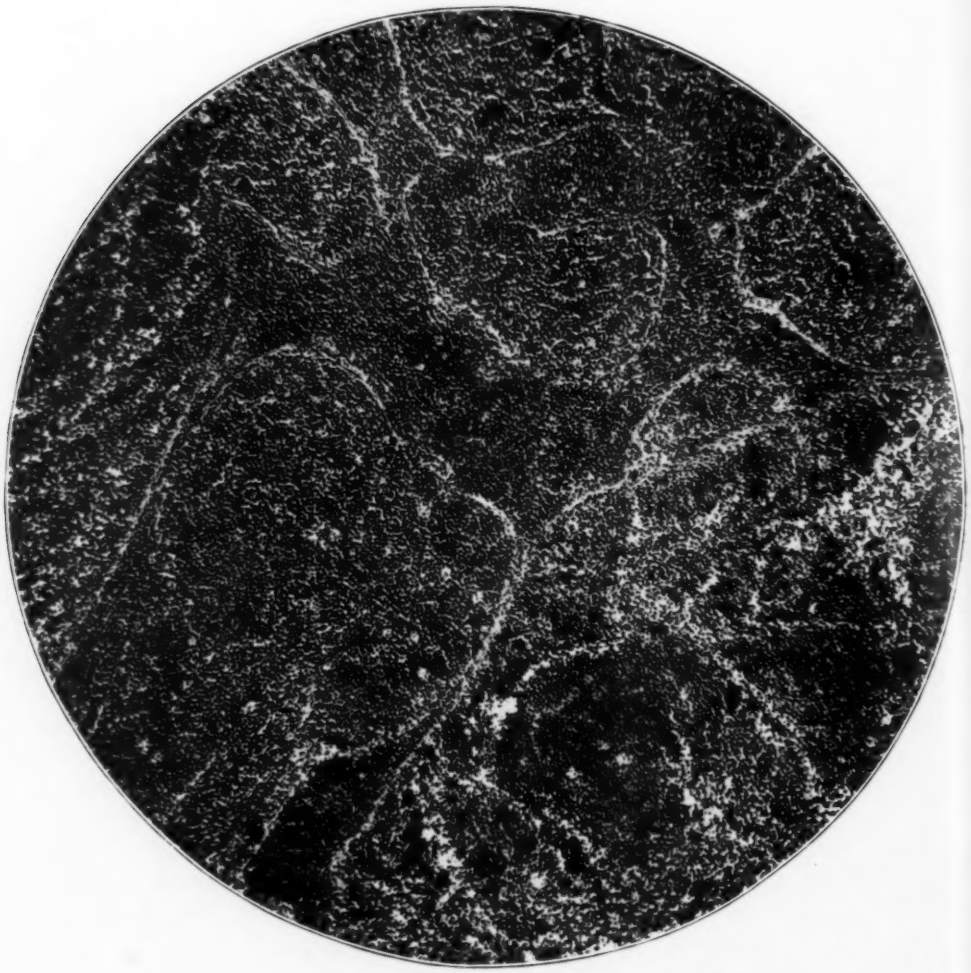

Fig. 1.—Low-power photomicrograph of the growth in the prostate, showing huge numbers of lymphocytes arranged in small islands, separated from one another by delicate strands of connective tissue.

REPORT OF CASE

History.—A man, aged 30, born in Denmark, and from whom a satisfactory history was not obtained, was admitted, March 22, 1918, and died April 16. The patient stated that, in the course of the seven weeks before admission to the hospital, he lost 20 pounds (9 kg.) in weight and that lately he had become

obstinately constipated. He likewise complained of a sharp pain in the region of the left kidney that radiated downward in the direction of the corresponding iliac fossa. He, moreover, stated that it was frequently necessary for him to void urine and that, on one occasion, he had great difficulty in passing it for a period of several days.

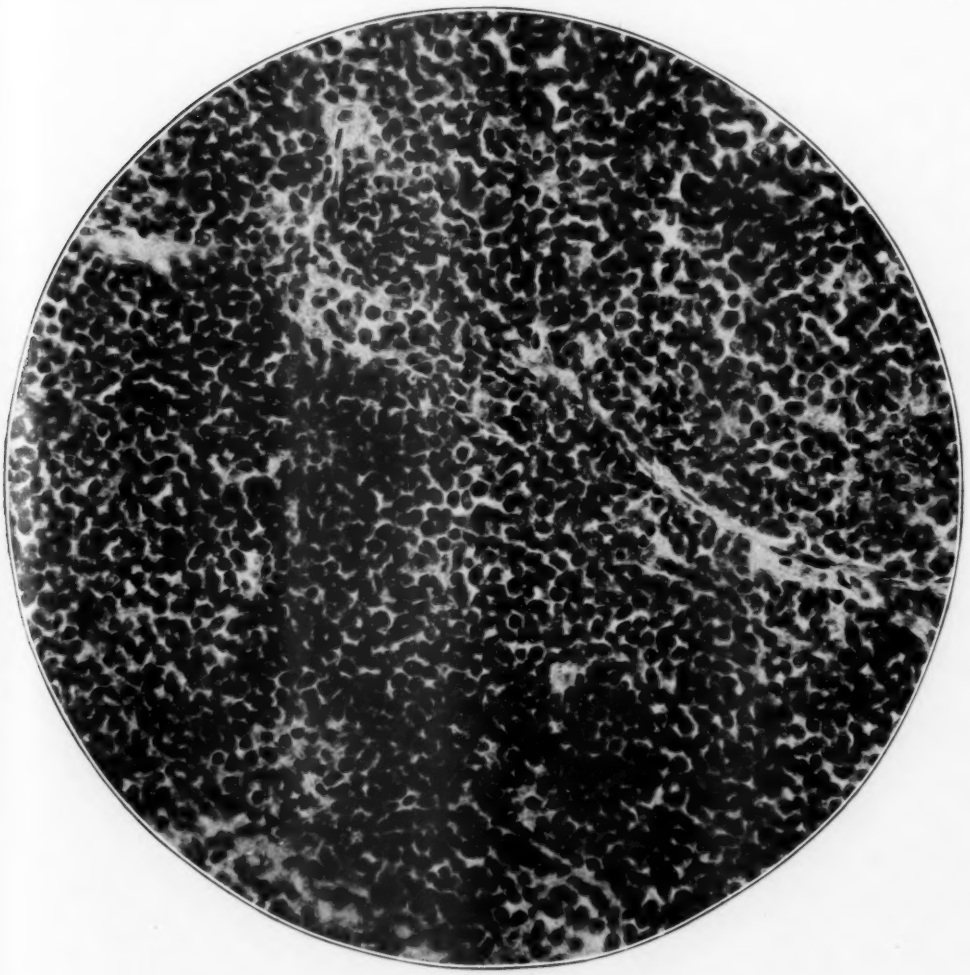

Fig. 2.—High-power photomicrograph of the prostatic growth.

Examination.—The urine revealed nothing abnormal other than a small number of pus cells. Roentgen-ray examination of the region of the left kidney failed to reveal the presence of a shadow suggesting a stone. The Wassermann reaction was negative with both antigens. The blood count showed 4,200,000 red cells; 70 per cent. hemoglobin; 12,800 white cells, of which 81 per cent. were polymorphonuclear leukocytes and 19 per cent. lymphocytes.

Four days after admission to the hospital, cystoscopy was attempted; but the instrument would not pass the prostatic portion of the urethra and the effort was abandoned.

Necropsy.—The body was 168 cm. in length. The region of the prostate was occupied by a growth that measured 9 cm. in diameter and 7 cm. in length. It surrounded the posterior urethra and was continuous with the lower third of the bladder, in which locality the walls of the bladder were thickened to the

Fig. 3.—Low-power photomicrograph of infiltrating hordes of lymphocytes in the cortex of the kidney.

extent of about 1 cm. and, on section, presented a pale, smooth, cream colored surface. The thickening of the bladder wall gradually disappeared as the viscus ascended into the pelvis, so that at the fundus the thickness was about normal. The bladder was dilated, measuring 12 cm. from trigon to fundus. The mucous membrane was swollen and presented numerous large and small,

velvety-looking, faintly reddish splotches. The prostatic growth presented, on section, a perfectly smooth, cream colored or whitish appearance. The substance was fairly friable. The seminal vesicles could be seen embedded in and compressed by the growth. Laterally, the growth invaded the psoas magnus muscle on both sides. The rectum was not involved. Strewn along the front of the spine on both sides, from the brim of the pelvis upward as far as the level of the pancreas, were numbers of large and small

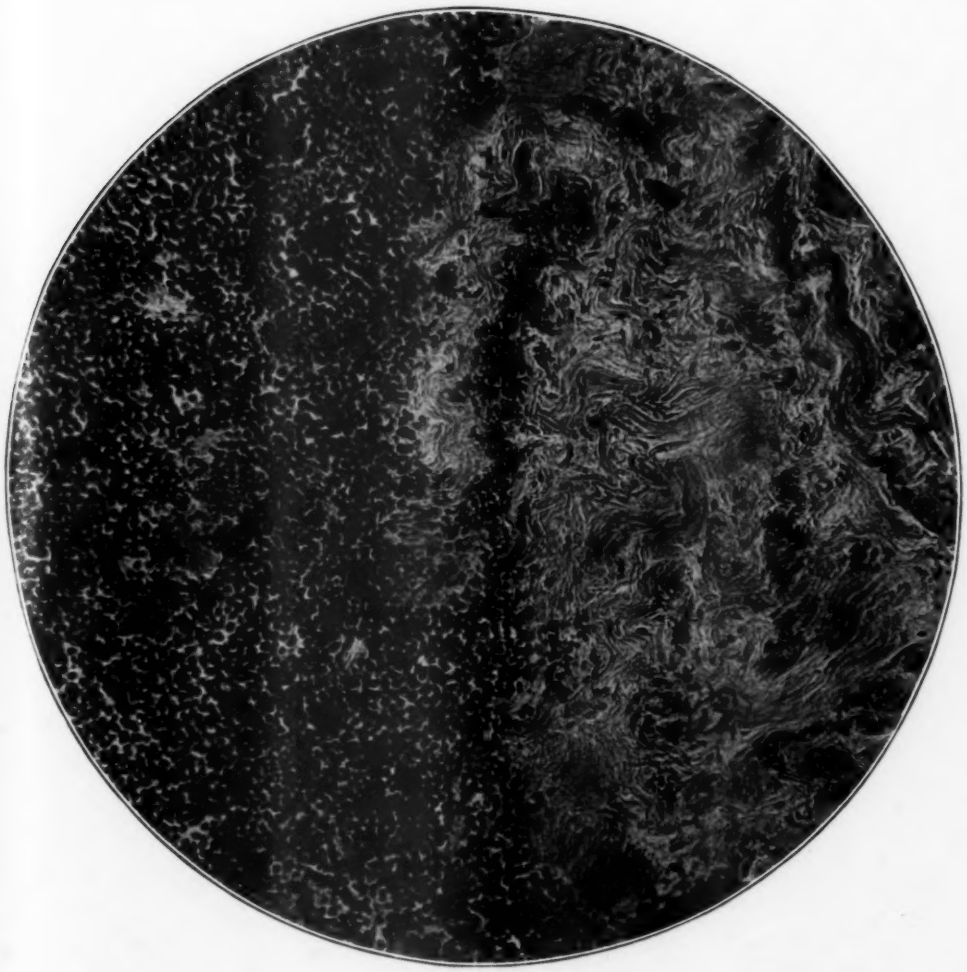

Fig. 4.—Low-power photomicrograph showing lymphosarcomatous infiltration of the walls of the ureter.

rounded, oval or flattened nodules, some of which were discrete, others fused, that presented, on section, much the same appearance, to the naked eye, as the tumor in the prostate. The testicles were unchanged. The left kidney was considerably enlarged. Projecting beneath the capsule were a dozen or more small cream colored elevations. Similar bodies were found deep in the medul-

lary portion near the pelvis. The upper third of the ureter was twisted and dilated by fluid. The ureters on both sides ran directly through the large masses of cream colored tumor tissue that lay at the side of the spine. The left suprarenal capsule was buried in tumor tissue of much the same general appearance as that in the prostate and elsewhere. The liver was enlarged. Beneath the capsule, and scattered over the cut surface, were large numbers of grayish or slate colored streaks or patches, between which the liver tissue was

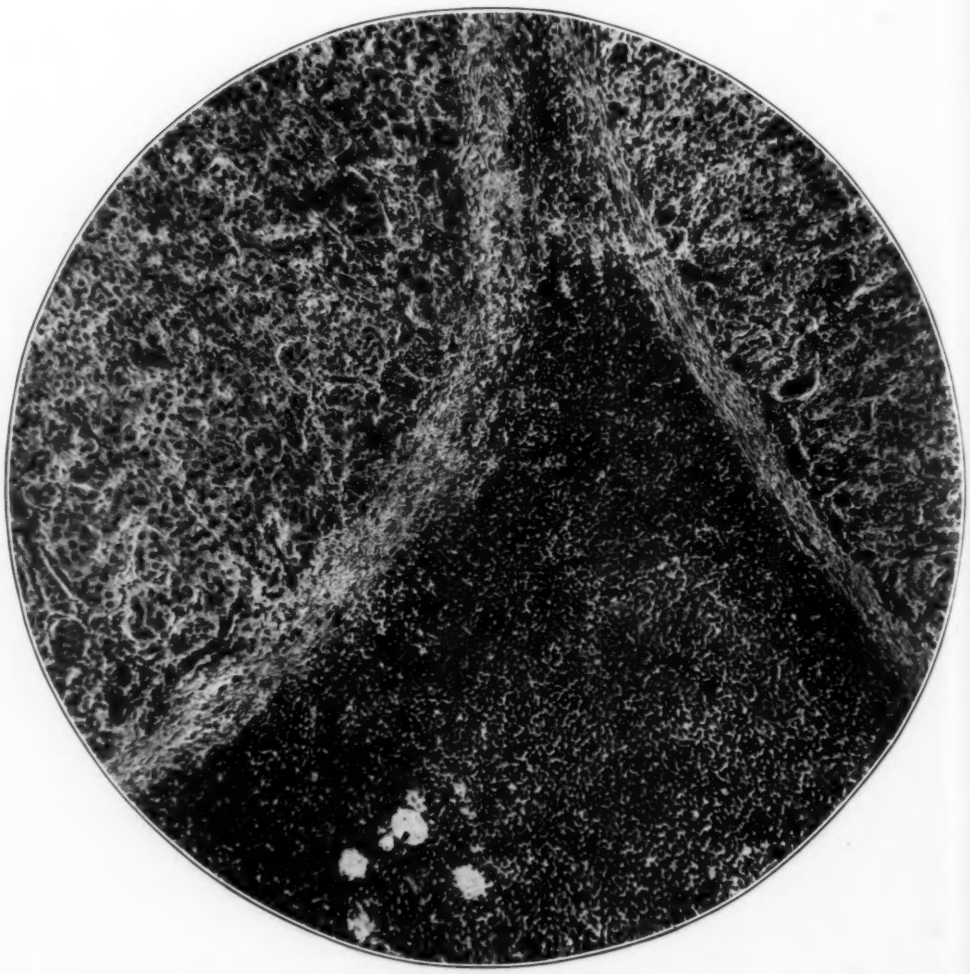

Fig. 5.—Low-power photomicrograph showing lymphosarcomatous infiltration of the tissues around the suprarenal capsule.

faintly brownish in color. Under the pleura on the left side, corresponding to the upper border of the fifth rib in the midaxillary line, was a small oval cream colored nodule. The pleura covering it was intact, as was the border of the rib against which it lay. The remaining organs showed nothing worthy of note in the present connection.

Anatomic Diagnosis. — This was: massive lymphosarcoma of the prostate, with extensive infiltration of both psoas magnus muscles, and compression of the seminal vesicles; bilateral neoplasmic infiltration of the retroperitoneal lymph nodes and of both kidneys, with dilatation of the ureters and periureteral neoplasmic infiltration; neoplasmic infiltration of the liver; solitary metastatic deposit in the left parietal pleura.

Fig. 6.—Low-power photomicrograph showing extensive lymphosarcomatous invasion of the perilobular connective tissues of the liver.

Histology.—Microscopic examination of the primary growth in the prostate revealed the presence of vast numbers of small round cells presenting the histologic characteristics of lymphocytes. For the greater part, the cells were growing diffusely, but, in places, they were arranged in small islands separated from one another by delicate strands of connective tissue (Fig. 1). Careful scrutiny of

microscopic sections from the original growth, as well as from its secondary deposits, failed to reveal the slightest indication of glandular formations. In other localities, the histologic structure of the tumor differed in no essential particular from that of the prostate, although insular formations were not to be seen, the cells growing diffusely.

Fig. 7.—Low-power photomicrograph illustrating neoplasmic destruction of the psoas magnus muscle.

COMMENT

The belief that tumors composed of lymphoid cells may arise in the prostate has been challenged on the ground that the prostate is a glandular organ whose fibromuscular stroma does not normally contain lymphocytic tissue. It is maintained that the small round-cell tumors of

the prostate are in reality carcinomas, the cells of which have become so altered as to resemble lymphoid cells, and that careful search will eventually disclose in some part of all such growths a return to the glandular arrangement by which these epithelial tumors of the prostate are characterized. I am unable to subscribe to this view. It has been shown that the prostate belongs to the so-called auxiliary lymphoid system, that is to say, that, with the suprarenal capsule, thyroid, lung, liver, kidney, testicle and other organs, the prostate normally contains interstitial lymphoid foci, which, in ordinary circumstances, are likely to be overlooked, but, in certain conditions of disease, are readily identified. There is no apparent reason why a tumor of lymphoid cells should not spring from such a focus in the prostate, nor, indeed, why any one or all of the other members of this group of organs should not react similarly. That they do not more commonly give rise to lymphocytic growths is to be explained by the fact that the lymphoid rests in them are numerically insignificant and that they are functionally dormant, undergoing hyperplasia only in unusual circumstances, whereas the follicles of the lymph nodes and spleen, tonsils, gastro-intestinal tract, etc., are geographically distributed in such fashion as to serve as filters, in which capacity they are constantly engaged in the process of compensatory regeneration and hence are frequently subject to disturbances of cell equilibrium.

THE PHYSIOLOGY OF PULMONARY EMBOLISM AS DISCLOSED BY QUANTITATIVE OCCLUSION OF THE PULMONARY ARTERY *

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Pulmonary embolism is a condition frequently encountered in clinical practice. The picture varies from symptoms produced by multiple small emboli lodged in the lung periphery, to symptoms resulting from massive blocking of one or both pulmonary arteries. In the case of small emboli, though severe chest symptoms often result, the patient usually recovers, while in massive blocking it is commonly believed that death ensues either immediately or within a few hours.

That massive pulmonary embolism may occur without disastrous effects on the general circulation was first shown by Lichtheim.[1] Two years later Welch [2] demonstrated that ligation of the circulation to one entire lung may be accomplished without effect on the systemic blood pressure—an observation confirmed later by Plumier [3] and also by Gerhardt.[4] Tigerstedt [5] corroborated and extended these observations, showing, by means of the stromuhr, that occlusion of half of the pulmonary circulation decreased only slightly the minute volume output of the left ventricle. Mann [6] supported this evidence by making intravenous injections of wax and clots into dogs until the pulmonary circulation was largely obstructed, without affecting either blood pres-

* From the Laboratories of Physiology, Harvard Medical School.

1. Lichtheim: Die Störungen des Lungenkreislaufes und ihr Einfluss auf den Blutdruck, Inaug. Dissert., Berlin, 1876.

2. Welch, W. H.: Zur Pathologie des Lungenödems, Virchows Arch. f. path. Anat. **72**:375 (March) 1878.

3. Plumier, L.: La circulation pulmonaire chez le chien, Arch. internat. de physiol. **1**:176, 1904.

4. Gerhardt, D.: Experimentelle Beiträge zur Lehre vom Lungenkreislauf und von der mechanischer Wirkung pleuritischer Ergüsse, Ztschr. f. klin. Med. **55**:195, 1904; Zur Lehre von der Hypertrophie des rechten Ventrikels, Arch. f. exper. Path. u. Pharmakol. **82**:122 (Oct.) 1917.

5. Tigerstedt, R.: Ueber den Lungenkreislauf, Skand. Arch. f. Physiol. **14**:259, 1903.

6. Mann, F. C.: Pulmonary Embolism: An Experimental Study, J. Exper. Med. **26**:387 (Sept.) 1917.

sure or respiration. Dunn,[7] in 1920, was able to inject 15 c.c. of starch into the jugular vein of goats anesthetized with ethyl carbamate (urethan), weighing from 20 to 30 kg., without producing any effect other than a pronounced dyspnea. Histologic examination of the lungs of Dunn's animals showed "that the degree of obstruction of the pulmonary arterioles is very great."

More recently Underhill,[8] employing, in addition, a Schafer[9] pulmonary cannula, has made somewhat similar observations on cats with both open and closed chests. He reports that under artificial ventilation (chest open) ligation of the left pulmonary artery causes an average rise in pulmonary arterial pressure of about 40 per cent.

The experiments cited show clearly that a large part of the pulmonary circuit may be blocked without causing a fatality. There is, however, no direct evidence as to the exact amount of obstruction required.

Our investigation was undertaken at the suggestion of Dr. C. K. Drinker in an attempt to observe the effect of graded pulmonary arterial occlusion upon the systemic and pulmonary blood pressures and to note possible variations in lung ventilation. The heart preparation which we employed was that recently devised by Drinker[10]—a preparation which affords easy access to, and control over, the heart and all the great vessels in a normally breathing animal, and permits experimentation in this region under conditions more closely approaching the normal than has heretofore been possible.

TECHNIC

The animals employed were cats; ethyl carbamate, in the majority of the experiments, being the anesthetic used. Because of the possible depressing effects of this drug upon the respiratory center, a few control experiments were performed under ether, but so far as the experimental results are concerned there was no essential difference.

The operative technic may be briefly summarized as follows: Under artificial respiration an oval area in the chest wall, immediately over the heart and roughly corresponding to its size, is removed, and hemostasis obtained. The pericardium is then incised longitudinally

7. Dunn, J. S.: The Effects of Multiple Embolism of Pulmonary Arterioles, Quart. J. Med. **13**:129 (Jan.) 1920; The effects of Multiple Pulmonary Embolism, J. Physiol. (Proc. Physiol. Soc.) **53**:5 (May) 1919.

8. Underhill, S. W. F.: An Investigation Into the Circulation Through the Lungs, Brit. M. J. **2**:779 (Nov.) 1921.

9. Schafer, E. S.: A Convenient Method of Recording Pulmonary Blood-Pressure, Quart. J. Exper. Physiol. **12**:133, 1918-1920.

10. Drinker, C. K.: A Useful Heart Method, J. Exper. Med. **33**:675 (June) 1921.

and the cut margins are sewed to the edge of the chest wall with interrupted sutures. The air imprisoned in the chest cavity is then aspirated and the pleural cavity rendered air-tight. Negative intrathoracic pressure having thus been restored, artificial respiration is stopped, and the animal is permitted to breathe naturally. In one instance an animal, prepared in this manner and maintained under complete anesthesia

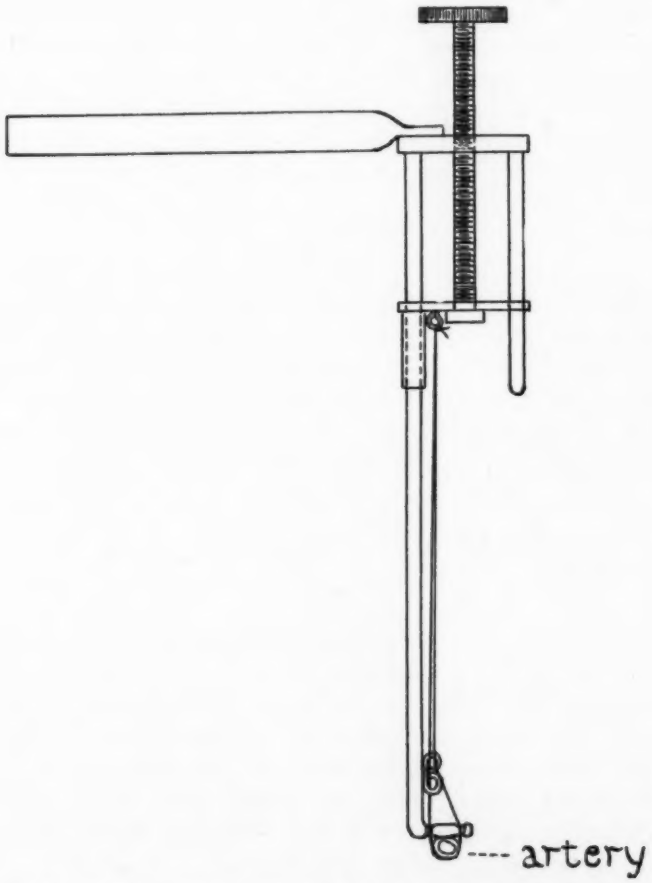

Fig. 1.—Clamp A, used for compression of the pulmonary artery and its left branch.

on a warm table, lived through the night and was used for experiment the next day—a period of more than eighteen hours.

As a rule, the pulmonary artery bifurcates just inside the pericardium, hence it is easy to slip a ligature around the left branch. Clamp A, illustrated in Figure 1, is then adjusted. Its right-angled base rests lightly against the artery, and the ligature is threaded into the clamp as shown in the illustration. Another ligature is then passed

around the whole pulmonary artery just above the bifurcation, thus allowing a sufficient length of the artery to permit insertion of the pulmonary cannula without impinging upon the pulmonic valves. In passing this ligature some sharp dissection is often necessary as the aorta and pulmonary artery are frequently bound firmly together. The pulmonary artery ligature is next threaded into a clamp similar to that controlling the left branch. This arrangement enables the operator, by the manipulation of a thumb screw, to compress the whole pulmonary artery, its left branch, or both, and, if he desires, to release the compression.

The pulmonic cannula [11] is then inserted into the main artery just below the angle formed as it bends posteriorly toward the hilus of the lungs. As a final step, a glass oncometer, covered with a very fine rubber dam, is slipped into place. A stitch, taken previously in the apex of the heart, is led through the oncometer tube and, with the aid of a needle, is passed through the rubber tube connected with the recording lever. The heart is thus held snugly in place within the bell of the oncometer. All of these adjustments are easily made after completing the operative procedure described and can be accomplished without the use of artificial respiration.

The tracheal cannula (Fig. 2) is then connected through the arms *A* and *B* (*C* and *D* being shut off) with the inspiratory and expiratory Tissot valves. The arms *C* and *D*, the inflow and outflow lines, respectively, connect with the artificial respiration pump. Arm *E* is tied into the trachea. When artificial respiration is in use, *A* and *B* are, of course, closed. The expired air of the animal escapes through a T-tube into the room or may be collected and accurately measured in a delicately balanced spirometer. Thus, the volume of respired air is easily determined at any time during the experiment.

The systemic blood pressure is recorded from the femoral or from the carotid artery in the usual way. Both systemic and pulmonary cannulas are connected with manometers by means of rubber tubing filled with 8 per cent. sodium citrate solution—the systemic cannula with a mercury manometer, the pulmonic cannula with a delicate membrane manometer. The respiratory rate is recorded by means of a pneumograph placed either on the upper abdomen or on the lower thorax of the animal.

Figure 3 illustrates the clamp (Clamp B) employed in producing graduated partial compression of the pulmonary artery. The bar *A-B* is passed beneath the artery, resting lightly against its inferior surface,

11. Swift, W. E.; Haggart, G. E., and Drinker, C. K.: A New Method for Measuring the Pressure in the Pulmonary Artery, J. Exper. Med. **36**:329 (Sept.) 1922.

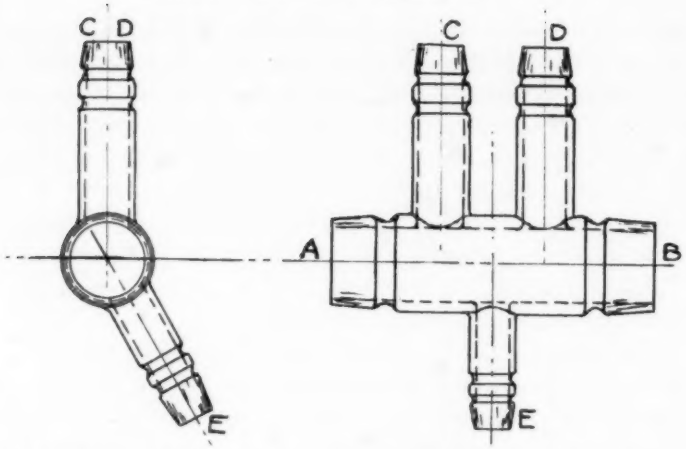

Fig. 2.—Tracheal cannula: cross-section and anterior view.

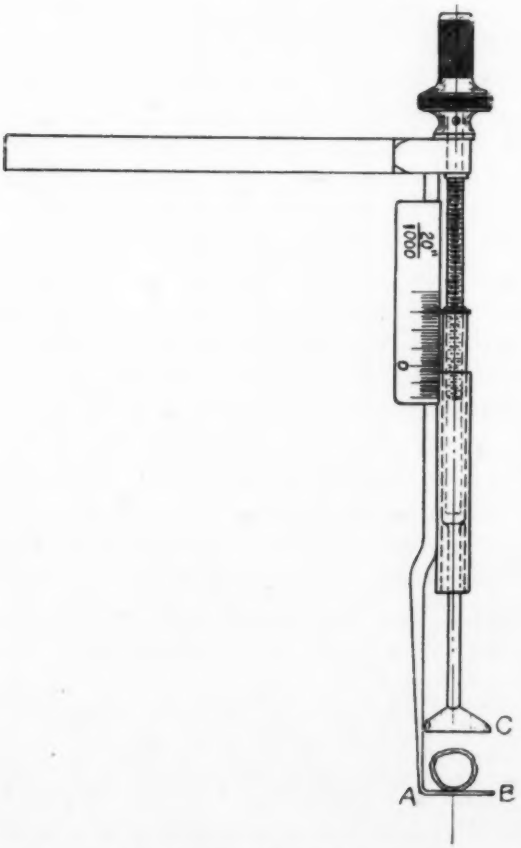

Fig. 3.—Clamp B, used for graduated partial compression of the pulmonary artery.

the vessel being placed in the center to allow for lateral expansion in both directions. The plunger *C* is then screwed down until it just touches the superior wall of the artery. Readings on the scale (which is adjustable) give the external diameter of the artery and the zero from which the compression readings begin.

At the end of each experiment, the plunger was screwed down until the artery was occluded. The corresponding scale readings were then noted and interpreted in terms of a curve developed from a series of calculations made from an elastic rubber tube. In developing the curve the rubber tube was compressed gradually, and at various points inked impressions of its end were stamped on paper. This resulted in a series of diagrams showing change in shape and area of the tube during progressive compression. A measurement of these diagrams made it possible to compute the area of the opening during any degree of compression by the formula, $O = \pi r^2 + 2rl$, in which O is the area of the opening at the point of compression; r, the radius of the lateral curvatures of either side of the compressed tube; and l, the transverse distance across the artery from the end of one curved edge to the beginning of the other. The calculations so obtained have been plotted in terms of percentage, the degree of compression against the percentage decrease in radius.

EXPERIMENTS

The left pulmonary artery was completely occluded sixteen times in a series of thirteen cats, for periods varying from three to fifteen minutes (with one compression of seventy-two minutes). Records were made of the resulting changes in systemic and pulmonic blood pressures, in the cardiac output and in the rate and volume of respiration. These changes are presented in Table 1 and may be summarized thus:

Blood Pressure in the Femoral Artery.—The average pressure before compression was 70.5 mm. of mercury, the range being from 44 to 110 mm. The application of compression was without immediate effect on the pressure level. Subsequently, and usually within the first minute of the experiment, the pressure altered somewhat. In nine of the twelve cases recorded, this alteration was a rise, but in no instance did it exceed a 14.2 per cent. variation.

Blood Pressure in the Pulmonary Artery.—In the fifteen cases in which this measurement was recorded, the average pressure preceding compression was 14 mm. of mercury, the range being from 9 to 23 mm. In all instances except one, in which no change occurred, there was an abrupt rise in pressure when compression was applied, but in only three cases was this rise greater than 33 per cent. (50, 83.3 and 100

TABLE 1.—Effect of Total Occlusion of the Left Pulmonary Artery on the Blood Pressure in the Systemic and Pulmonary Arteries, on the Heart, and on the Rate and the Volume of Respiration

Experiment	Duration of Occlusion, Min.	Femoral Blood Pressure in Relation to Compression, Mm.			Pulmonic Blood Pressure in Relation to Compression, Mm.			Heart Findings in Relation to Compression			Respiration in Relation to Compression				
								Dilatation During	Biventricular Minute Volume, C.c.		Tidal Air (in 3 Min.), C.c.			Rate per Min.	
		Before	During	After	Before	During	After*		Before	During	Before	During	After	Before	During
1	3	..	....	..	..	....	..	Slight, until compression release	680	518	734	1,193	567	25	20
2A	6	104	110-104	104	19	24	24	Slight, prolonged	...	...	1,910	2,075	1,938	35	30
2B	6	108	108-105	108	23	26-24	19	Slight, temporary	210	100, 260, 210	1,938	2,130	1,9.0	30	35
3	72	62	62 (30′)†	..	14	15-14 (30′)	..	None (30′)	900	800 (30′)	5,990	(7,830 (15′) (5,770 (20′)	.....	150	240
4A	3	50	51	50	12	14-12	12	Slight, temporary	720	720	2,348	2,941	2,518	40	45
5	5	74	66-68	73	18	20-19	18	None	435	360	1,800	2,250	1,900	50	55
6	2	..	.....	..	13	15-12	13	None	540	540	.....	.....	.....	30	30
8	11	60	64-66	..	12	16-15	..	None	374	331	1,467	1,962	.....	30	30
9	14	62	64	..	17	19-15	..	None	468	468	1,260	1,701	.....	30	35
10A	8	44	46-45	..	12	22-17	..	Slight, temporary	653	691	.....	.....	.....	30	35
10B	4	..	.....	..	12	14	..	Severe, permanent‡	562	546	.....	.....	.....	..	..
11	5½	50	52	..	12	18	..	None	672	672	.....	.....	.....	30	35
12A	5	66	74	74	12	16	14	Slight for 10 seconds	846	800	2,022	2,440	2,300	38	40
13 (ether)	2	110	113-110	108	9	18	..	None	826	825	1,900	2,050	.....	35	35
After vagal section:															
4B	3	..	.....	..	14	14	..	None	638	570	2,755	2,990	.....	17	22
12B	5	56	64	..	11	13-12	..	None	900	900	1,912	1,318	.....	..	..

* This measurement was recorded whenever possible, but in eight cases compression was not released until further experimentation rendered readings of no comparative value.

† Numbers in parenthesis indicate minutes after beginning of compression at which observation was made.

‡ This compression was made on a heart previously weakened by a temporary occlusion of the entire pulmonary artery.

TABLE 2.—Effect of Complete Compression of the Pulmonary Artery, Following Permanent Occlusion of the Left Pulmonary Artery, on Blood Pressure in the Femoral and Systemic Arteries, on the Heart and on the Rate and Volume of Respiration

Experiment	Duration of Compression, Sec.	Femoral Blood Pressure in Relation to Compression, Mm.			Pulmonic Blood Pressure in Relation to Compression, Mm.			Heart Findings in Relation to Compression				Respiration: Variety During and After Compression
								Dilatation		Minute Volume, C.c.		
		Before	During	After	Before	During	After	During	After	Before	After	
9	32	52	16	61	15	37	11	Severe	Recovery	689	437-648	Respiration became irregular and, if the degree of compression was sufficient, ceased even though the clamp was loosened
10A	50	44	19	32	14	31	14	Moderate*	Partial recovery	814	690-800	
10B	50	..	..	..	12	31	13	Severe	No recovery	484	369	
11	27	46	17	22	12	44	..	Severe	No recovery	706	237	

* Compression was less complete in this instance.

per cent., respectively). In the remaining twelve records the average change was only 16.9 per cent., or 2.7 mm. The average for the fifteen determinations was 29 per cent. During compression the tendency seemed to be toward a return to the control pressure, especially in those cases in which the shift had been least extensive. Compression release was usually without marked results in decreasing the pulmonary arterial pressure.

Cardiac Dilatation.—In nine of the sixteen instances, the oncometer record showed no cardiac dilatation whatever at any stage of the compression. In six of the remaining seven a very slight dilatation occurred immediately upon the application of compression, but in all except two of these—in which dilatation lasted until compression was removed—the heart returned to its control size (in from ten seconds to two and one-half minutes) before the pulmonary block was released. The only case of severe permanent dilatation occurred in a heart which had been previously weakened by a temporary occlusion of the entire pulmonary artery. In this instance compression release did not cause the heart to return to its normal size.

Cardiac Minute Volume.—In fifteen instances, the biventricular output before compression ranged from 210 to 900 c.c., the average being 628 c.c. per minute delivered in 168 strokes of approximately 4 c.c. each. In six of the experiments, no change whatever took place during compression. In another seven, there was a permanent decrease in volume that averaged 11.6 per cent. and in no case exceeded 23.8 per cent. This decrease was accomplished more often by a diminished output for each beat than by a diminution of rate, though the latter sometimes occurred. In one experiment, there was a minute volume increase of 5.8 per cent. In another (Experiment 2 *B*, Table 1), we obtained a temporary decrease in minute volume accompanying some degree of cardiac dilatation, with a return to normal after a compensatory period of increased output.

Tidal Air.—With the exception of two unusual cases in which the figures of 734 and 5,990 were obtained, the three-minute volume of tidal air before compression, with vagi intact, ranged from 1,260 to 2,348 c.c. The average in the ten cases without vagal section was 2,137 c.c. In every instance, collections taken during the period of compression showed an increased tidal air. This increase varied from 7.9 to 62.6 per cent., was below 12 per cent. three times, above 35 per cent. once, and averaged 25.7 per cent. Collections made from one to three minutes after compression release showed a return toward the control figures in every case. In twelve of the fourteen cases in which the respiratory rate was determined, the average rate before compression was 33.6 per minute, and after compression, 35.4 per minute

—an increase of 5.4 per cent., which seems insufficient to account for the increased tidal air without premising an accompanying increase in respiratory depth.

Table 2 illustrates the striking reaction secured by adding a brief but almost complete occlusion of the pulmonary artery to permanent compression of its left branch. The results vary with the severity of the compression applied; but it will be noticed that while the femoral blood pressure falls sharply to about one third of the control level—an average variation of 63 per cent.—the pulmonic pressure rises from 121 to 267 per cent.; that the heart dilates severely and usually permanently, its minute volume decreasing from 15 to 66 per cent., and that respiration becomes irregular and stops. If the application is brief enough, the systemic pressure will again rise, respiration will recommence, and the animal will live for some time.

Figures 4 and 5 record typical results secured by compression of (*a*) the left pulmonary artery and (*b*) the entire pulmonary artery. Both of these tracings are taken from Experiment 9, the details of which are given herewith.

REPORT OF EXPERIMENT

EXPERIMENT 9.—Oct. 23, 1922, a cat, weight 2.7 kg., at 9 a. m., was given 6.75 gm. of ethyl carbamate by stomach tube. At 11:05 a. m., the operation was finished with chest tight and with ligatures placed around the left pulmonary artery and around the pulmonary artery just mesial to its bifurcation. The ligature clamps and the cardiometer were adjusted; a cannula was inserted into the pulmonary artery, and arrangements made for recording the rate of respiration and femoral blood pressure. At 11:41 a. m., a control collection showed the tidal air to be 1,260 c.c. in three minutes.

At 11:50 a. m., the left pulmonary artery clamp was tightened and the entire blood supply of the left lung thereby suddenly and entirely obstructed.

The result is illustrated in Figure 4. Tracing 1 is the oncometer cord, and reveals alteration neither in the size, rate nor stroke delivery of the heart. Tracing 2 represents the abdominal respiration and shows an increase in rate following compression of from 30 to 35 per minute. Tracing 3 shows a femoral blood pressure of 62 mm. of mercury, which rises after compression to a sustained level of 64 mm. Tracing 4 is the pulmonary blood pressure, and, in this instance, presents on compression a mean pressure rise of from 17 to 19 mm. of mercury. Marks on the base line of this tracing signal events in the experiment. Tracing 5 is the time clock marking one-second intervals.

An air collection made at this juncture gave a three-minute total of 1,701 c.c., an increase over the control of 34.5 per cent.

At 11:56 a. m., after a five-minute occlusion of the left pulmonary artery, the ligature about the pulmonary artery was tightened. The occlusion, which was almost complete, was loosened at the end of thirty-one seconds.

The results of the latter procedure are illustrated in Figure 5. The tracing numbers here have the same indications as those in Figure 1. Mark 1 shows the application of pressure; Mark 2, its removal. It will be noticed that:

(*a*) The heart dilated sharply and the stroke output decreased markedly (689 to 437 c.c.). One minute after compression was released, the dilatation was becoming less, and the minute volume was compensatorily great (792 c.c.). In another minute the cardiac size was as in the control, and the minute volume had returned to normal (648 c.c.).

(*b*) Respiration became irregular, ceased entirely, and did not begin again until compression had been removed for thirty-seven seconds.

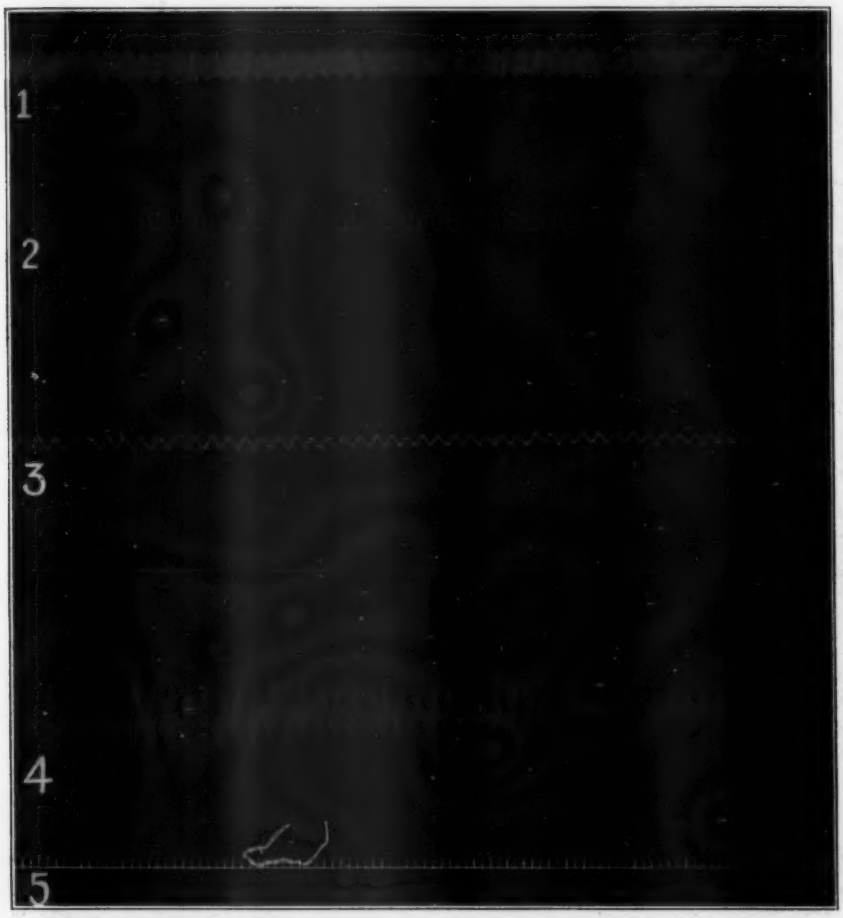

Fig. 4.—Record showing absence of any reaction following compression of the left pulmonary artery (Experiment 9): Tracing 1, oncometer record of heart volume; Tracing 2, respiration; Tracing 3, systemic blood pressure; Tracing 4, pulmonic blood pressure; Tracing 5, one-second intervals. Mark 1 on the base line of Tracing 4 signals the application of compression.

(*c*) Very shortly after the ligature was tightened, the femoral blood pressure fell quite abruptly from 52 to 16 mm. of mercury. At this level it continued until compression was released, when it rose to its former level in approximately seventy seconds.

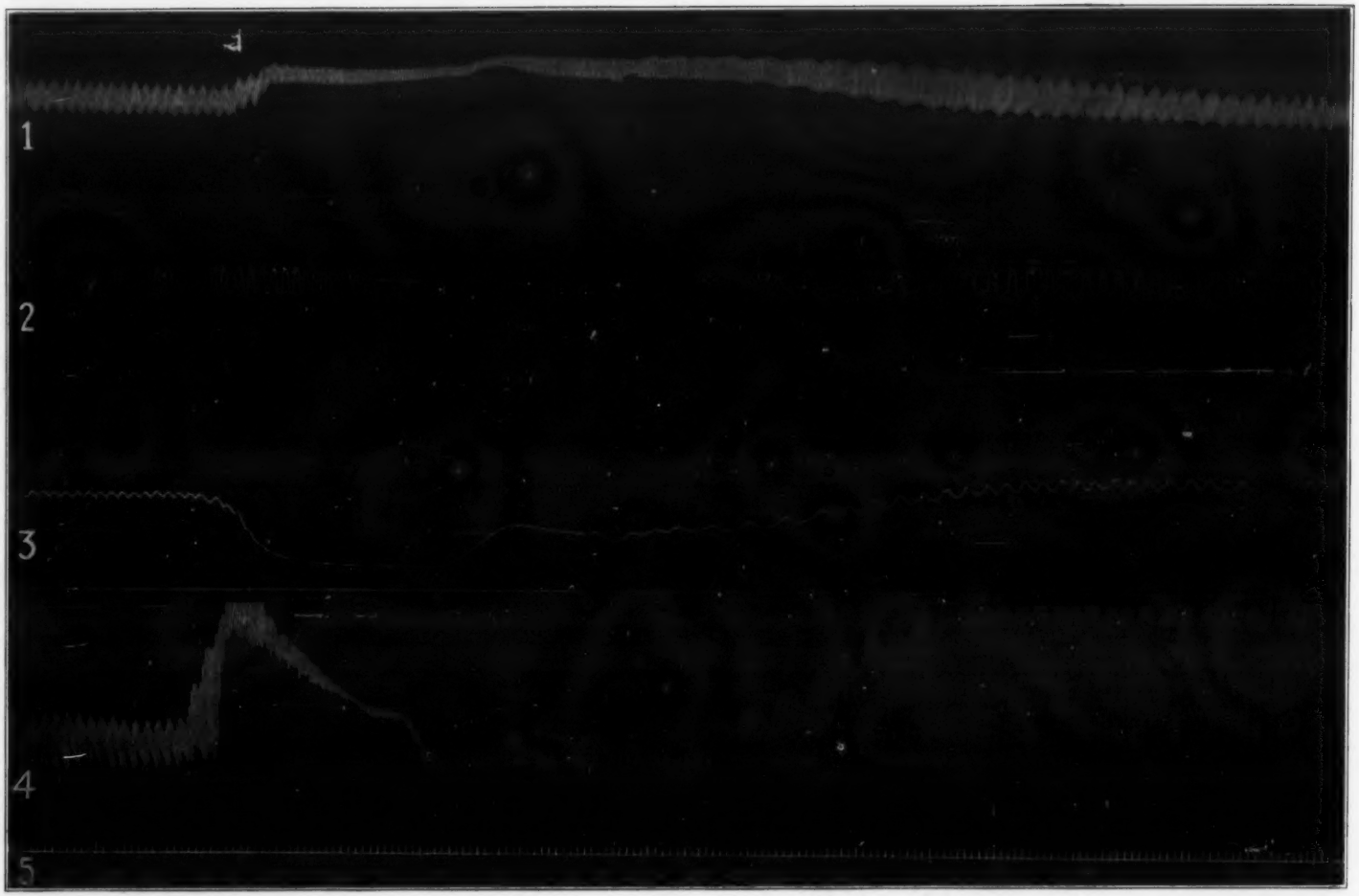

Fig. 5.—Record showing effect of total compression of the pulmonary artery (Experiment 9): Tracing 1, oncometer record of heart volume; Tracing 2, respiration; Tracing 3, systemic blood pressure; Tracing 4, pulmonic blood pressure; Tracing 5, one-second intervals. Marks on the base line of Tracing 4 signal application and release of compression.

(*d*) The pulmonary blood pressure rose very suddenly from 15 to 37 mm. of mercury, the up-stroke being limited by the interference of the systemic base line. Before compression was removed it had fallen to 18 mm. of mercury, owing probably to failure of the right heart as a result of coronary anemia. After removal it fell to 11, and ninety seconds later it again rose to 28 mm. of mercury, where it remained.

At 12:03 p. m., a collection showed that the three-minute tidal air had increased to 2,108 c.c. At 12:04, compression was removed from the left pulmonary artery. At 12:08, the tidal air had fallen to 1,913 c.c. At 12:09, the animal was bled to death from the femoral and pulmonary arteries. Examination showed the ligatures to be in the positions indicated.

In one experiment in this series (Experiment 3), forty-five minutes after pressure was applied, the heart had not dilated, the pulmonary arterial pressure

TABLE 3.—Effect Upon the Systemic and the Pulmonic Arterial Pressure of Graduated Partial Compression of the Pulmonary Artery

	Systemic Blood Pressure, Mm. Hg	Pulmonic Blood Pressure, Mm. Hg	Cardiac Size	Biventricular Minute Volume, C.c.	Respiratory Rate per Minute
Experiment 19					
Without compression.....	62	12	Normal	504	30
At 60% compression.....	58	13	Slight dilatation	504	30
At 62-66% compression.....	18	14	Sharp dilatation	77	30
Experiment 20					
Without compression.....	60	17	Normal	858	24
At 60% compression.....	60	19	Slight dilatation	780	32
At 62-66% compression.....	12	20	Sharp dilatation	252	40 (irregular and stopped)
Experiment 21					
Without compression.....	54	17	Normal	806	30
At 60% compression.....	44	17	Dilatation	672	30
At 62-66% compression.....	12	34	Dilatation	101	Irregular and stopped
Experiment 26					
Without compression.....	48	10	.....	...	48
At 60% compression.....	38	11	.....	...	48
At 62-66% compression.....	12	12	.....	...	Irregular and stopped

remained at its control level, the femoral pressure had only fallen 12 per cent., and though the cardiac output had decreased 30 per cent. the tidal air was but 10 per cent. less than the control. Shortly after this, the lung ventilation began to decrease quite rapidly. With this change the femoral pressure fell; the heart dilated, and in an hour and twelve minutes the cat died.

These and similar experiments show not only that there are no immediate results of vital consequence from occlusion of the left pulmonary artery but also that secondary changes are extremely slow in making their appearance.

Our work with graduated compression of the pulmonary artery in four cases is summarized in Table 3, and Figure 6 gives a tracing from Experiment 19. Mark 2 on the record is the point where occlusion reached 60 per cent.; and mark 3, where it reached 65 per cent. In

these four experiments the pressure levels were unchanged at 55 per cent.; a slight reaction occurred at 60 per cent., and the end-point, marking the rapid onset of death, occurred on the average at 64 per cent.—the range being from 62 to 66 per cent. In the remaining three cases this end-point occurred with this degree of occlusion: 64 per cent., 55 per cent. and 52 per cent. Figure 7 records the strik-

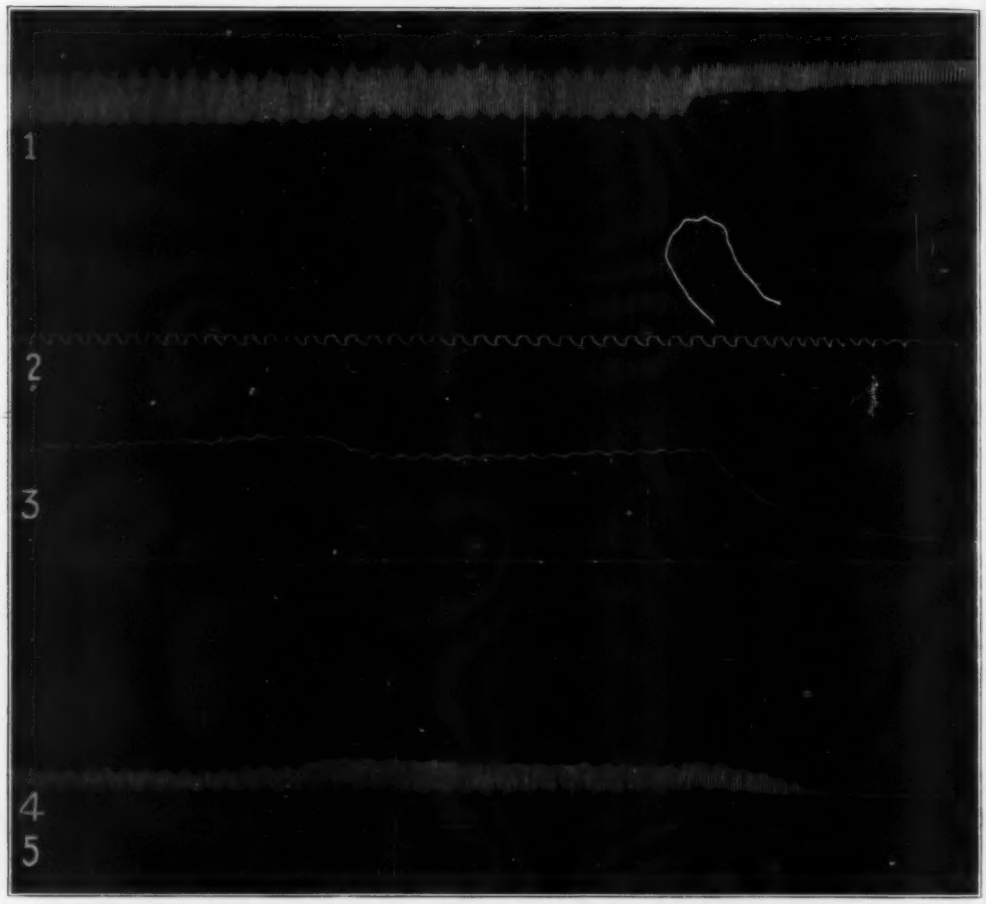

Fig. 6.—Record showing effect of graduated partial compression of the pulmonary artery (Experiment 19): Tracing 1, oncometer record of heart volume; Tracing 2, respiration; Tracing 3, systemic blood pressure; Tracing 4, pulmonic blood pressure; Tracing 5, one-second intervals. Mark 2 on the base line of Tracing 4 signals compression of the artery amounting to 60 per cent. occlusion. Mark 3 signals 65 per cent. occlusion—the point of heart failure in this animal.

ing results obtained in Experiment 26. The pulmonary artery was occluded three times with exactly the same amount of compression applied in exactly the same manner, and in each instance failure

occurred when 55 per cent. of the artery was shut off. Following release of the clamp after the first and second compressions, the heart diminished in size, and the systemic blood pressure again mounted to a level slightly below the control. At this point the procedure was again repeated, but following release of the clamp for the third time the respiration failed to begin, the heart remained dilated, and the animal died.[12]

COMMENT

These experiments were undertaken to determine the reactions of the greater and lesser circulation and of the respiration to partial and to complete pulmonary arterial block, a condition, as here presented, quite analogous to pulmonary embolism in man. The technic described has provided experimental conditions more closely approaching those existing in the normal animal than it has heretofore been possible to secure. In consequence, we believe the results may be regarded as indicative of the circulatory reaction to be expected in a case of pulmonary embolism.

The experiments fall into three groups: (*a*) those in which the left pulmonary artery alone was compressed, (*b*) those in which the whole artery was suddenly occluded, and (*c*) those in which the whole artery was obstructed by graduated partial occlusion. The latter work was done with the idea of ascertaining to what extent the total pulmonary circulation could be encroached upon before signs of general cardiac and systemic failure became evident.

(*a*) *Results of Left Pulmonary Artery Compression.*—In these experiments certain reactions, or, better, lack of reactions, were observed. To begin with, dilatation as measured by the oncometer occurred in the heart itself in only seven out of sixteen cases. In four of these it was very slight, and recovery occurred during the compression. In two it was also slight, but recovery did not take place until the block was removed. In the remaining case the dilatation was sharply marked, and without recovery. In this instance, however, the entire artery had been compressed and released early in the experiment, and the heart was undoubtedly damaged. The remaining experiments revealed no dilatation of the heart whatever. In six out of fifteen cases, moreover, there was no alteration during compression of the minute volume output of the heart. Of the other nine experiments, all except one show only a slight decrease (Table 1). So far, therefore, as the entire removal of one half of the pulmonary circulation is concerned, the effect upon the heart is essentially nil. This observa-

12. In this experiment, between Mark 7, when conditions in the animal were not seriously abnormal, and Mark 8, when cardiac failure occurred, the lumen of the pulmonary artery was narrowed by 0.036 sq. cm.

tion is in entire accord with that of Underhill,[8] who sums up his experience thus: "The healthy heart, therefore, can accommodate itself without difficulty to sending the same volume of blood through one lung only in a given time, as it previously sent through both."

As to the effect upon the pulmonary arterial pressure (Table 1), it will be observed that in every instance except one the pressure rose, usually rather abruptly, the degree of the rise varying in height, according to experimental conditions, but averaging about 29 per cent. During compression the tendency was toward a return to the control level. Thus, the rise in pressure in the pulmonary artery following an *embolus* of its left branch was quite distinct, and yet not sufficient to embarrass the heart action. It is of interest to contrast these findings with those obtained by compression of the pulmonary veins. In the latter experiments, the pulmonary arterial pressure rises sharply and to a height which frequently is twice that recorded when the left artery is compressed. Results of experiments of this type will be published later.

The effect on systemic blood pressure caused by sudden occlusion of the left pulmonary artery is shown in Table 1. In one out of twelve cases, compression produced no change. In nine of the experiments there was an average rise of 4.2 mm.; while in the remaining two an average fall of 5.5 mm. occurred. It is clear, therefore, that halving the pulmonary circuit has little or no effect upon the general systemic circulation.

The respiration, under this procedure, shows a constant change. The number of inspirations usually increases to a slight extent and their depth is greater (direct observation). The total ventilation shows a definite and, at times, a marked increase, the average rise being 25.7 per cent.

The cause of this hyperpnea raises an interesting point. It is doubtless due in part to the fact that one lung alone must now aerate the blood. Dunn[7] is of the opinion that this hyperpnea is probably "brought about by reflex action, initiated by stimulation of sensory nerve endings in the lungs." He cites experiments in which, with the vagi cut, extensive starch embolism was produced in goats. The respiratory rate, however, remained quite unaltered.

To investigate this point, we carried out experiments in which the left pulmonary artery was clamped before and after vagal section. The resultant circulatory changes were essentially those already described, but there was a decided difference in the effect upon the ventilation. After cutting both nerves, in one instance a previous increase of 25 per cent. was reduced to 9 per cent., while in another experiment an increase of 20 per cent. was replaced by a reduction in ventilation of 30 per cent.

These findings confirm those of Dunn [7] and make additionally justifiable the assumption that the vagus nerves are evidently an important link in causing the hyperpnea following pulmonary embolism. It is also worth while to comment, in this connection, on the definite evidence presented of a "very considerable reserve of vascular area in the lungs." In our experiments the function of one entire lung is wiped out, and yet the general circulation is well maintained—at least, as Dunn adds, "for the resting animal."

In summary, we may say that an animal under ethyl carbamate or ether anesthesia can bear a sudden stoppage of blood flow in the left pulmonary artery—a condition analogous to that of embolism of the vessel in man—without any sustained effect other than some increase in the respiratory ventilation and a rise of approximately 29 per cent. in pulmonary arterial pressure.

(*b*) *Results of Total Pulmonary Artery Compression.*—In this group of experiments in which the entire pulmonary circulation was suddenly blocked off, very marked changes in the respiration and circulation were observed (Table 2). When the pulmonary circulation is entirely blocked, there results an immediate dilatation of the heart and a rapid diminution of its output. The pulmonary arterial pressure rises sharply—on the average between 121 and 267 per cent.—and then falls. This occurs before the clamp is removed, presumably because of right ventricular failure dependent on insufficient coronary flow which results from a lowered systemic pressure. The systemic blood pressure falls rapidly and, unless the compression is immediately removed, continues to drop until it reaches zero.

It is worth while pointing out that, following this compression, the fall in systemic blood pressure, as indicated by the sloping curve obtained (Fig. 5), is not abrupt but is, on the whole, a rather gradual decline. This occurrence is probably best explained as being due to the gradual feeding of the blood present in the lungs at the time of the arterial obstruction to the left heart through the mechanical act of respiration. The final failure of systemic pressure is thus delayed in proportion to the volume of blood present in the lungs.

The effect of compression of the pulmonary artery upon respiration is immediately noted. The breathing becomes irregular, soon stopping altogether, and usually fails to begin again even when the compression is promptly released. Total sudden occlusion of the lesser circulation, even when present only a few seconds, evidently, therefore, entails such severe results that the animal is seldom able to recover. This fact still holds even though, with the compression released, artificial respiration is begun and epinephrin given into the left side of the heart.

Clinically these facts are borne out by the findings of Petrén,[13] who, from an analysis of a series of patients dying of pulmonary embolism, is of the opinion that following embolic block of one pulmonary artery death does not always supervene, whereas in those persons suffering from even temporary occlusion of the whole artery, the result is invariably fatal.

In brief, we believe the sequence of events leading to death from pulmonary embolism to be a rise of pressure in the pulmonary artery, dilatation of the right ventricle and a fall in systemic blood pressure, resulting in malnourishment and failure of the right heart. The respiratory center fails following the drop in systemic blood pressure.

(*c*) *Results of Graduated Partial Compression of the Pulmonary Artery.*—Up to this point the observations reported are those made following occlusion, first of one-half and then of the whole pulmonary artery. To complete the study of quantitative occlusion of this artery, it was our aim to determine how much of it could be occluded before there appeared any signs of cardiac failure, fall in blood pressure, etc.—in other words, to discover to what degree the whole pulmonary circuit might be blocked off before symptoms developed which were at all comparable to those resulting from complete embolism of the artery. To obtain such data, it was first necessary to devise means by which it would be possible to narrow the lumen gradually and, the diameter of the artery being known, to measure accurately at any moment the degree of the occlusion. This would enable us to determine the exact amount of compression applied when the recording manometers first gave evidence of an embolic effect. Clamp B, illustrated in Figure 3, fulfilled these requirements.

The operative procedure was the same as that previously described, except that in the majority of the cases a ligature tied around the left pulmonary artery was substituted for Clamp A. Experimental results with the left branch patent were essentially the same as those noted below. When Clamp B was in position, the plunger was gradually screwed down, starting with a distance equal to four scale divisions, and then progressively increasing the compression, one unit at a time —a compression of one-fiftieth of an inch. Following each increment of compression, sufficient time was always allowed to make sure that there was no delayed after-effect.

In each experiment, this gradual narrowing of the pulmonary arterial stream continued without obvious effect until a large part of the total pulmonary artery was closed. At this juncture there was a definite endpoint. Indeed, so sharply was it defined that further clamp

13. Petrén, G.: Studien über obturierende Lungenembolie als postoperative Todesursache, Beitr. z. klin. Chir. **84**:606 (May) 1913.

closure of one-fiftieth of an inch precipitated an immediate reaction. When this endpoint was reached the heart dilated sharply, with a corresponding decrease in minute volume output; the systemic pressure fell rapidly at first and then more gradually, while the pulmonic pressure rose abruptly. Respirations became irregular and soon ceased entirely. The percentage occlusion of the artery which produced such a reaction in four of these experiments is strikingly similar—66, 65, 64, and 62 per cent., or an average of 64 per cent.

In the remaining cases the reaction occurred at 55 and 52 per cent. occlusion. The first of these is of particular importance and is illustrated in Figure 7. The fact that exactly the same degree of occlusion could be provided, three successive times, and in each instance would bring about an abrupt and, if unrelieved, fatal reaction in the animal is of great interest and is the subject of further investigation in this laboratory. Many factors must take part in inducing this type of cardiac failure. As the pulmonary artery is narrowed, the amount of blood reaching the left ventricle finally becomes less and less. Systemic blood pressure falls and with it the pressure in the coronary vessels. At the same time the heart dilates, the muscle fibers are placed under increased tension and probably require additional oxygen for adequate contraction, instead of the reduced amount with which they are provided, owing to the lowered systemic blood pressure. Furthermore, it seems probable that as dilatation advances the cardiac capillaries may suffer mechanical closure or reduction in lumina so that a further factor is added to the vicious cycle which arterial obstruction has initiated.

Postmortem Findings.—In those experiments in which occlusion of the left pulmonary artery was maintained, a distinct, though not marked, difference in the appearance of the two lungs was noted. The left lung, from which the blood was shut off, was definitely paler than the right, the latter presenting a faint pink blush, quite noticeable when contrasted with normal lungs. This was particularly well demonstrated in the experiment on the animal in which the clamp was maintained in place for more than an hour. In these findings we disagree with Underhill, who notes that the blocked lung, chest closed, is always more congested, while in an open chest experiment the opposite is the case.

It is interesting to note that when the pulmonary artery clamp was removed, following a period of compression, and blood was again allowed to flow through the lung, postmortem examinations showed the lung congested, a marked generalized reddish color—at times even a brilliant salmon hue—being visible. The lung felt soggy, and a thin bloody fluid oozed freely from the cut surface. On section, the alveoli

Fig. 7.—Record showing effect of repeated (three times) graduated partial compression of pulmonary artery (Experiment 26): Tracing 1, respiration; Tracing 2, systemic blood pressure; Tracing 3, pulmonic blood pressure; Tracing 4, one-second intervals. The marks on the base line of Tracing 3 signal application of units of compression, Mark 8 in each series indicating 55 per cent., occlusion—the point of heart failure in this animal.

were engorged with red corpuscles. There was some edema. This change we consider due to capillary damage, possibly resulting from a local alkalosis due to deprivation of carbon dioxid.

CONCLUSIONS

1. In the method described, it is possible, in the normally breathing animal to produce partial or complete block of the pulmonary artery, the condition as here brought about being analogous to pulmonary embolism in man.

2. Clamping the left branch of the pulmonary artery causes an immediate rise in pulmonary pressure, averaging about 29 per cent., and such a procedure causes no significant change in the general systemic pressure nor any significant variation in size of the heart, heart rate or heart output. Ventilation, as measured by a spirometer, is, however, increased approximately 25 per cent., a moderate hyperpnea resulting.

3. Following total pulmonary occlusion a severe and immediate reaction sets in. The heart dilates quickly and to a marked degree, the minute volume output becoming materially less. The pulmonary pressure rises sharply—121 to 267 per cent.—and then gradually falls toward zero. The systemic pressure begins to fall immediately and does not recover, while respirations become irregular and shortly thereafter cease altogether.

4. Until from 52 to 66 per cent. of the pulmonary circulation is cut off there is no significant variation in the general circulatory condition of the animal. The point at which failure occurs is sharply defined, since beyond this endpoint a circulatory collapse is precipitated by a minute increase in the arterial obstruction, but if this is not applied no untoward change results.

STUDIES ON THE CALCIFICATION OF CALLUS*

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ST. LOUIS

In a previously published article, I [1] reported the results of experiments which I had performed to determine the effect of variations in the blood phosphorus on the rate of calcification of callus in fractures. These experiments were made on the basis of reactions in vitro and with recent work on rickets as a background; and they were entirely negative. Continued interesting reports on experimental rickets have stimulated the present attempt to accelerate the calcification of fracture callus through the application of the factors that will influence the calcification of bone in that condition.

Workers on the vitamins lay stress on the fact that no good is to be expected from any superabundance of these substances in the dietary intake. Deficiency alone is of significance. In the presence of conditions brought on by deficiency, an addition to the diet of the particular vitamin concerned will result in a return to normal. The fact that the ingestion of cod liver oil under such circumstances will result in a rapid deposition of calcium in calcium-deficient bones [2] suggests the possibility that this power to influence the deposit of calcium might extend beyond the abnormal rickety state, in spite of the general rule mentioned above. It was hoped that the effect might be the same in a condition in which there is equally a functional demand for calcium, namely, in the presence of a soft fracture callus.

It has been shown that, in animals fed on diets deficient in fat-soluble A, the deficiency state could be prevented from developing by

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*From the Department of Surgery, Washington University School of Medicine.

1. Lehman, E. P.: Studies in Inorganic Blood Phosphate, J. Biol. Chem. **48**:293 (Oct.) 1921.

2. Shipley, P. G.; Park, E. A.; McCollum, E. V.; Simmonds, N., and Parsons, H. T.: Studies on Experimental Rickets, II. The Effect of Cod Liver Oil Administered to Rats with Experimental Rickets, J. Biol. Chem. **45**:343 (Jan.) 1921. McCollum, E. V.; Simmonds, N.; Becker, J. E., and Shipley, P. G.: Studies on Experimental Rickets, XXI. An Experimental Demonstration of the Existence of a Vitamin Which Promotes Calcium Deposition, Bull. Johns Hopkins Hosp. **33**:229 (June) 1922.

exposure to direct sunlight.[3] In the present series of experiments, the effect of sunlight was also tested in the healing of fractures in the normal rat.

In a third type of experiment, the effect on fracture callus of each of these two influences in animals rendered somewhat rickety was also studied. Diet 3143 of McCollum and his associates [4] was chosen. This diet will produce marked rickets in young rats in from thirty-five to forty days and will not produce xerophthalmia. It contains "proteins of good quality (about 33 per cent.) and about twice the calcium necessary for optimal growth and nutrition. It is, however, low in fat-soluble A and below the optimum in its content of phosphorus." [5] Sunlight and cod liver oil, to the amount of 2 per cent. of the weight of the diet, will each protect against the development of rickets. The study of the healing of fractures in rickets was not intended; it was hoped only that, if the effect of these influences on callus was not demonstrable in normal rats, they might be shown to affect callus favorably if there was established a tendency toward the withdrawal of calcium from the bones. For this reason, the preliminary period of feeding was limited to twenty-four days, and the animals were all killed at thirty-eight days. Such animals did not show marked anatomic changes at death; but they were distinctly below the normal in weight, and their bone repair, as will appear, was less strong. Unfortunately, the material for microscopic study of the epiphyses and callus was lost.

TECHNIC

The white rat was employed because it is the animal for which the foregoing facts have been standardized. Each experiment was conducted with a single litter, divided into control and experimental animals. They were fed on the standard laboratory diet of corn, carrots, meat and dog biscuit, unless otherwise stated. The left humerus was used for experimental fracture and the right for control.

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3. Hess, A. F., and Unger, L. J.: The Cure of Infantile Rickets by Sunlight, J. A. M. A. **77**:39 (July 2) 1921. Hess, A. F.; Unger, L. J., and Pappenheimer, A. M.: Experimental Rickets in Rats, III. The Prevention of Rickets in Rats by Exposure to Sunlight, J. Biol. Chem. **50**:77 (Jan.) 1922. Park, E. A.; Powers, G. F.; Shipley, P. G.; McCollum, E. V., and Simmonds, N.: Studies on Experimental Rickets, XIV. The Prevention of the Development of Rickets in Rats by Sunlight, J. A. M. A. **78**:159 (Jan. 21) 1922. Clark, J. H.: The Physiological Action of Light, Physiol. Rev. **2**:277 (April) 1922.

4. McCollum, E. V.; Simmonds, N.; Shipley, P. G., and Park, E. A.: Studies on Experimental Rickets, VIII. The Production of Rickets by Diets Low in Phosphorus and Fat-Soluble A, J. Biol. Chem. **47**:507 (Aug.) 1921.

5. McCollum, E. V.; Simmonds, N.; Shipley, P. G., and Park, E. A.: Studies on Experimental Rickets, XV. The Effect of Starvation on the Healing of Rickets, Bull. Johns Hopkins Hosp. **33**:31 (Jan.) 1922.

The method of studying calcification by the roentgen ray is not subject to even approximately accurate quantitative interpretation. For that reason, a method of measuring the breaking and bending strength of the bones was employed. It is assumed in this method that the rigidity and strength of the callus is proportional to the calcium deposited in the cellular matrix. The apparatus illustrated in Figure 1, adapted from that used by Allison and Brooks,[6] was employed. The bone to be studied was placed in the grooves (*C*) of the side bars (*B*), within the yoke (*D*). The bone was accurately adjusted in exactly the

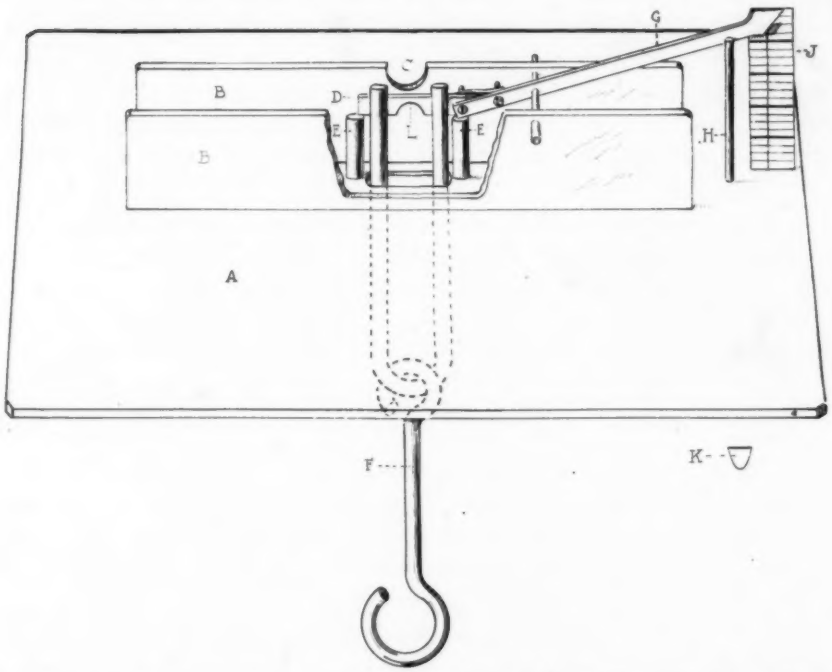

Fig. 1.—Apparatus for measuring bending and breaking strength of normal and fractured bones: *A*, base plate with orifice for yoke; *B*, *B*, side bars; *C*, groove for bone (a corresponding groove exists on the side bar broken away to show mechanism); *D*, yoke, with groove to fit over bone; *E*, *E*, stop posts; *F*, hook for sand bucket; *G*, bend-recording lever (ratio of 1:4); *H*, guide pin for lever; *J*, scale; *K*, cross-section of yoke at *L* to show edge where weight is applied to bone, rounded to prevent cutting of callus.

same relation to the side bars and the yoke in each instance. Sand was then added slowly to a light bucket, suspended from the hook (*F*), until breaking occurred. Bending was indicated by movement of the lever (*G*) on the dial (*J*) in a ratio of 4 to 1. The amount of weight necessary to bend or break was recorded by weighing the sand bucket.

6. Allison, Nathaniel, and Brooks, Barney: Bone Atrophy, Surg., Gynec. & Obst. **33**:250 (Sept.) 1921.

A series of twenty-six rats was employed in working out the details and accuracy of this technic. These preliminary experiments demonstrated that the method of placing the bone in the breaking machine is all-important. As can be seen by the sketches, the humerus of the rat is marked by a prominent ridge, which extends from the head, about halfway down the bone. In the control bones from each rat, the yoke was centered exactly at the base of this spur and the latter was made to lie in the horizontal plane. When so placed, results were sufficiently uniform to lie within biologic limits. At the conclusion of the experiments, three normal rats were studied as a final check on the method. In each of these, the breaking strengths of both humeri were measured. The results are shown in Table 1. Breaking of the normal bones always occurred sharply, without preliminary bending.

The problem of obtaining uniformity in measuring the strength of the callus of healing bone proved more difficult. Fortunately, it was

TABLE 1.—BREAKING STRENGTHS OF RIGHT AND LEFT HUMERUS IN NORMAL RATS

Rat	Right, Gm.	Left, Gm.	Percentage of Difference
1	1,920	2,080	8
2	2,300	2,190	5
3	4,080	3,800	7

found that by practice the bones can be broken at the same point in vivo in the vast majority of attempts. The rats were anesthetized with ether and the bones snapped with the fingers. Fortunately, also, it was found that the position assumed by the fragments is fairly uniform if breaking occurs at the usual site. The lower fragment rides up along the spur of the bone, and angulation of about 15 degrees, or less, occurs. Bones healing in this position can be placed under the yoke uniformly, and the weight adjusted to the apex of the callus. The only significant variation occurs when the angulation is more acute, and the bone shifts position so that the force is applied at the inner point of the angulation. Such readings have been discarded.

Figure 2 shows the method of recording each experimental fracture, and examples of the results obtained. In each, the line across the bone represents the line of fracture and the arrow the angulation of the lower fragment. Numbers 15 and 17 represent the typical fractures, with typical displacement. Number 18 is an example of an unusual line of fracture at the usual site, with displacement so typical as to permit adjustment in the apparatus in such a manner as to bend and break the callus in the usual way. Numbers 16 and 28 show typical positions of the fragments and angulation slightly in the reverse direction. These

also were susceptible of accurate measurement. Number 21 is an example of an unsatisfactory result, which was discarded. Here the fracture is at an abnormal site, and sharply angulated.

When adding weight to healing bones, there is a slow bending, through an arc on the scale of 1 to 1.5 cm. This bending is uniform at first. Later, it becomes more rapid, until it finally ends in the complete rupture of the callus.

After a total experience with about seventy-five rats, in all of which both humeri were studied, the method appeared sufficiently accurate to be of value for the ends desired.

The preliminary studies revealed that the most favorable stage in healing for rats of the age used (from 7 to 11 weeks, at the time of breaking the bones) was at fourteen days.

EXPERIMENTS

All of the experiments were, therefore, performed in accordance with the following general scheme: The litter was divided into control

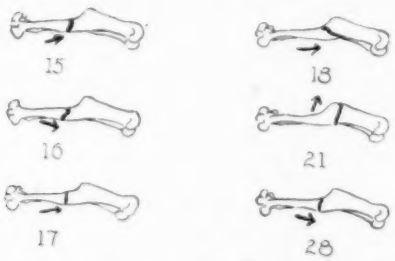

Fig. 2.—Typical and atypical lines of fracture and angulation. Compare text.

and experimental animals. The latter group was redivided in the different experiments, as indicated below. If dietary modifications were to be made, these were carried out on the experimental animals for the specified time, at the conclusion of which the entire litter was anesthetized and the left humerus of each animal was fractured. If no dietary modification were to be made, the breaking was done at the corresponding age. The experimental animals were then subjected, for a period of fourteen days, to the various procedures to be detailed. At the end of this time, the entire litter, control and experimental animals, were killed with ether. Both humeri were dissected out, with care not to disturb the integrity of the bone or of the callus, and the breaking strength of each was measured. The line of fracture and the angulation were recorded at the same time on diagrams, such as those reproduced in Figure 2.

As there was marked variation in animals of the same litter in the absolute breaking strength of the undamaged bone, expressed in grams,

the results can better be expressed in ratios, by dividing the breaking strength of the healing bone by the breaking strength of the undamaged bone. This method has given results of fair uniformity in the control animals of each litter, as shown in Table 2. The greatest variation within one litter is seen to be 0.10 in Litter B. It is to be noted here that the animals of Litter F, in which the most rapid healing occurred, were younger by two weeks than those of any of the other litters. The high reading in Litter C was on a single rat.

Six litters were used, totaling fifty-two rats. Of these, eighteen animals, including an entire litter, were discarded because of failure to obtain a satisfactory fracture. In the discarded litter, in which the rats were two weeks younger than the youngest of the others, the breaking point in the bone occurred near the head; there was no displacement, and union was too far advanced at fourteen days to make the results comparable.

TABLE 2.—RESULTS WITH CONTROL ANIMALS

Litter	Age in Weeks at Breaking Bone	Rat 1	Rat 2	Rat 3	Average Per Litter
D	11	0.58	0.56	0.55	0.56
F	7	0.73	0.76	0.70	0.73
A	9½	0.67	0.64	....	0.64
B	9½	0.56	0.47	0.57	0.53
C	9	0.73	....	....	0.73
		General average, 12 rats.....			0.64

Control Rats: Strength of fractured bone expressed in percentage of strength of normal bone.

When sunlight was to be employed, the rats were placed in the direct rays without interposition of glass during most of each afternoon, at least four hours daily and often six.

PROTOCOLS

Percentages represent the relation of the breaking strength of the callus to that of the normal bones, as in Table 2. Diet 3143 wherever it occurs in these protocols, refers, as elsewhere in this paper, to the diet devised by McCollum and his associates.[7]

I. ON NORMAL DIET

LITTER D.—Age at breaking of bones, 11 weeks; age at measuring strength of callus, 13 weeks.

Controls: 3 rats; average, 0.56.

Sunlight (2 weeks): 3 rats; average, 0.53.

Normal Diet Plus Cod Liver Oil: 3 rats; average, 0.54.

7. Footnotes 4 and 5.

LITTER F.—Age at breaking of bones, 7 weeks; age at measuring strength of callus, 9 weeks.

Controls: 3 rats; average, 0.73.

Sunlight (10 days): 3 rats; average, 0.62.

II. ON ABNORMAL DIET

LITTER A.—Age at beginning of dieting, 6 weeks; age at breaking of bone, 9½ weeks; age at measuring strength of callus, 11½ weeks.

Controls (normal diet throughout): 2 rats; average, 0.65.

Controls (Diet 3143 from age of 6 weeks): 2 rats; average, 0.54.

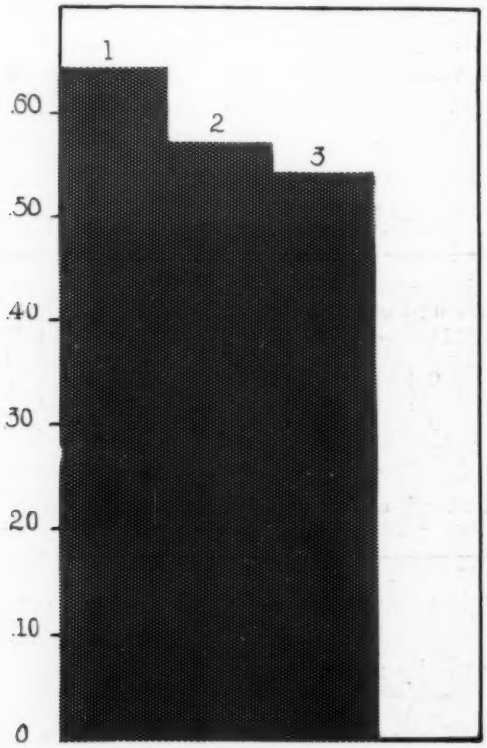

Fig. 3.—Consolidated averages of experiments under normal dietary conditions: Column 1, normal controls: 12 rats, 0.64; Column 2, normal diet plus sunlight: 6 rats, 0.57; Column 3, normal diet plus cod liver oil: 3 rats, 0.54.

Sunlight (for 2 weeks immediately following fracture; Diet 3143 from age of 6 weeks): 1 rat, 0.63.

Cod Liver Oil (Diet 3143 from age of 6 weeks; cod liver oil added after fracture): 1 rat, 0.47.

LITTER B.—Age at beginning of dieting, 6 weeks; age at breaking of bones, 9½ weeks; age at measuring strength of callus, 11½ weeks.

Controls (normal diet throughout): 3 rats; average, 0.53.

Controls (Diet 3143 from age of 6 weeks): 2 rats; average, 0.52.

Sunlight (for 2 weeks immediately following fracture; Diet 3143 from age of 6 weeks): 1 rat, 0.55.

Cod Liver Oil (Diet 3143 from age of 6 weeks; cod liver oil added after fracture): 1 rat, 0.51.

Litter C.—Age at beginning of dieting, 5½ weeks; age at breaking of bones, 9 weeks; age at measuring strength of callus, 11 weeks.

Controls (normal diet throughout): 1 rat, 0.73.

Controls (Diet 3143 from age of 6 weeks): 3 rats; average, 0.53.

Sunlight (for 2 weeks immediately following fracture; Diet 3143 from age of 6 weeks): 1 rat, 0.38.

Cod liver Oil (Diet 3143 from age of 6 weeks; cod liver oil added after fracture): 1 rat, 0.32.

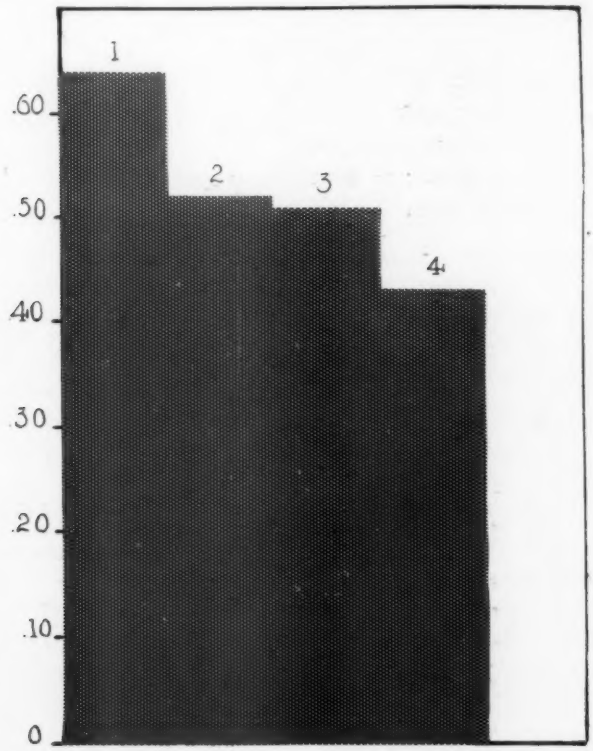

Fig. 4.—Consolidated averages of experiments under abnormal dietary conditions: Column 1, normal controls: 12 rats, 0.64; Column 2, controls, Diet 3143: 7 rats, 0.53; Column 3, Diet 3143 plus sunlight: 3 rats, 0.52; Column 4, Diet 3143 plus cod liver oil: 3 rats, 0.43.

COMMENT

A study of these protocols reveals that in no instance did the addition of cod liver oil to the diet of rats, either on a normal diet or on a rickets producing diet, result in a greater strength of the callus. In two litters, A and C, the callus of control rats on a rickets producing diet was somewhat less strong than in the normal controls, that is, from 0.54 to 0.65 and from 0.53 to 0.73; but cod liver oil did not tend to correct this defect in any instance.

Further, in the rats on a normal diet, sunlight had also no effect in accelerating the calcification of callus. In the rats on Diet 3143, in one series (Litter A), the effect of sunlight seemed favorable; but it must be remembered that the greater strength of the callus in the rat exposed to sunlight as opposed to that in the dieted control animal, that is, from 0.63 to 0.54, is within the limits of variation of the method as expressed in Table 2.

When, considering the averages of each litter as a coordinate unit, we consolidate these results, we obtain the graphic representations shown in Figures 3 and 4.

CONCLUSIONS

1. Exposure to sunlight and the addition of cod liver oil to the diet exert no measurable accelerating influence on the calcification of fracture callus in normal rats.

2. The same factors are equally nonoperative under experimental conditions in which such favorable effects might be expected to be intensified.

A STUDY OF DIVERTICULUM FORMATION IN THE APPENDIX*

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The subject of diverticulum formation in the appendix has apparently aroused the interest of but few writers in Europe and America. It seems rather astonishing in these days of experimental investigation of almost every conceivable subject that this interesting, if relatively unimportant, process has never, so far as I can ascertain, been produced or studied experimentally. Hypotheses have been based on morphologic observations of the appendix itself, together with the application to the appendix of a few experiments made in the large and small intestine. The results are unsatisfactory because some of them, reported by earlier observers, either could not be reproduced, or produced entirely different and contradictory results, when repeated by others.

It seems, therefore, that this condition deserves further investigation, particularly so inasmuch as I have become convinced during the past year that the condition is not nearly so infrequent as the few reported cases would lead one to believe. When, coincidentally with the change in the method of examining the appendixes in the laboratory of surgical pathology at the Presbyterian Hospital, we found five examples of diverticulum formation in the appendix in one year, whereas in the ten preceding years there was a record of only one appendix with a diverticulum, the inference is justifiable that many cases must have been overlooked. It was formerly our custom to make two or more transverse sections of the appendixes sent for examination, until the discovery of a case of multiple diverticula aroused our interest in making a careful search for this condition, and we began to section all the appendixes longitudinally in a plane passing through the mesenteric and antimesenteric borders. Four more appendixes came to our attention in this way, none of which were suspected or observed by the surgeons who removed them.

The questions which naturally arise in connection with diverticula of the appendix are: (1) Have they any clinical significance? (2) What are their morphologic characteristics and what are the factors which lead to their formation? (3) Is there any way in which they may be diagnosed clinically? (4) Can their formation be prevented, or, if they

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*From the Laboratories of Surgery at the College of Physicians and Surgeons, Columbia University, and the Laboratory of Surgical Pathology, Presbyterian Hospital, New York. Drawings by Alfred Feinberg.

have formed, is any treatment indicated? Most of those who have written about diverticula of the appendix have concerned themselves almost exclusively with the second of these questions, while a few have speculated about the first. This paper will deal very largely with the second question.

The discussion which follows is based on (*a*) the study of seven appendixes with diverticula, six of which were removed operatively at the Presbyterian Hospital, New York, and one at necropsy at the Bellevue Hospital by Dr. B. Morgan Vance and kindly given to me by him for study; (*b*) the reexamination of a large number of appendixes removed operatively at the Presbyterian Hospital during the last eleven years, for certain morphologic and pathologic characteristics which seemed to have some bearing on the formation of diverticula; (*c*) certain experiments on dogs, the results of which are suggestive in attempting to determine some of the etiologic factors; (*d*) a study of all the literature to which I could obtain access, with the desire both to learn what other investigators have observed and deduced and to ascertain from the photographs and drawings reproduced whether certain morphologic peculiarities which are apparent in the series reported in this paper were also present in other appendixes.

Before attempting to analyze the factors which seem to favor the formation of diverticula of the intestinal tract in general and of the appendix in particular, it will be pertinent to define what is understood by the term and to review briefly the hypotheses which have been advanced up to the present to explain them.

DESCRIPTIVE DEFINITION, STATISTICS AND HYPOTHESES OF DIVERTICULUM FORMATION

Most writers are agreed that diverticula in the appendix are "evaginations," "protrusions," "herniations," etc., of the mucosa, muscularis mucosae and submucosa through defects in the muscular coat. They are all said to be of the "acquired" variety, in contradistinction to the congenital diverticula which may be found in the intestinal tract, such as Meckel's diverticulum, in the walls of which are to be found all the coats which are present in the wall of the intestine. Such congenital diverticula have never been noted in the wall of the appendix.[1] They may be single or multiple. They are most commonly found along the mesenteric border, bulging out between the leaves of the mesenteriolum. Less commonly, they occur along the antimesenteric border and at the tip; very rarely elsewhere. In regard to frequency,

1. Krabbel: Divertikelbildung bei Appendicitis, Beitr. z. klin. Chir. **80**: 121, 1912.

MacCarty and McGrath [2] report seventeen in 5,000 appendixes (0.34 per cent.), Konjetzny [3] found two in 1,000 cases (0.2 per cent.), Moschcowitz,[4] four in 1,500 (0.26 per cent.) and Mertens [5] in 106 necropsies found two appendixes with diverticula, and in twenty-eight appendixes removed at operation, one diverticulum (together, 2.23 per cent.). If we take the five cases (Cases 1 to 5, inclusive) found in the one year, from July 1, 1921, to June 30, 1922, during which time 264 appendixes were removed at the Presbyterian Hospital, the percentage is 1.89.

The diverticula usually range in diameter from 3 to 5 mm. Rarely, if ever, do true mucosal lined diverticula reach a larger diameter than 1 cm., although mucoceles may reach a much greater size. They are evenly divided between males and females. The youngest patient apparently was Wilkie's,[6] a 15 year old girl; while MacCarty and McGrath [2] found one in a patient 64 years old. Usually, they have been found in appendixes acutely or chronically inflamed; occasionally, they have been found at necropsy or in patients operated on for some unrelated trouble. Reference to the accompanying statistical table will reveal that the cases reported here conform to these observations without any important variations, except a morphologic one which will be referred to later.

Of the acquired type, two groups are recognized: (1) those that are said to occur through the defects in the muscular coats which are found along the mesenteric and antimesenteric borders of the appendix, and through which pass the vessels which supply the submucosa and mucosa, and (2) those which occur through defects in the muscular coats which are results of acute inflammatory processes. Thus, there may be an intramural abscess which destroys the muscularis but which heals without perforation, leaving a scar in the muscularis through which the protrusion may subsequently occur; or there may be perforation with protrusion of the mucosa at the same time and, subsequently, subsidence of the acute inflammatory process without removal of the appendix. The last process may lead to the formation of an external mucocele, i. e., a subserous or intramesenteric collection of mucus in a cavity lined

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2. MacCarty, W. C., and McGrath, B. F.: Clinical and Pathologic Significance of Obliteration, Carcinoma and Diverticulum of the Appendix, Surg. Gynec. & Obst. **12**:211 (March) 1911.

3. Konjetzny: Zur Pathologie und Klinik der erworbenen Wurmfortsatzdivertikel, München. med. Wchnschr. **56**:2251, 1909.

4. Moschcowitz, E.: The Pathologic Diagnosis of Diseases of the Appendix, Ann. Surg. **63**:697 (June) 1919.

5. Mertens: Falsche Divertikel, etc., Mitt. a. d. Grenzgeb. d. Med. u. Chir. **9**:743, 1902.

6. Wilkie: Carcinoma of the Appendix Causing Diverticula of the Appendix and Acute Appendicular Obstruction, Brit. J. Surg. **8**:392 (April) 1921.

with connective tissue which communicates with the lumen of the appendix by a tract lined with mucous membrane extending through the muscularis.[7]

All of the foregoing statements are based on morphologic observations. The diverticula have been seen, and, as they were all accompanied by defects in the muscularis, it was assumed that muscular defects must precede their formation. The only muscular defects which could be found were those through which the vessels passed and those which were the result of damage following infection; ergo, the diverticulum must take place through these defects.

In order to explain why protrusions of mucous membrane occur through these defects in some appendixes and not in others, various hypotheses have been advanced. Most of them were used in an attempt to explain the occurrence of diverticula in the intestine, but they have been applied to those occurring in the appendix as well.

Klebs[8] states that, in obese people, collections of fat about the vessels as they pass through the wall tend to shove the muscle bundles apart, thus increasing the diameter of the defect and increasing its potential weakness. Graser[9] suggests that the chronic distention of the blood vessels as they pass through the muscular coats tends to push aside the muscle bundles, in this way increasing the diameter of the defect.

Practically all of the authors who attempt to account for the force which tends to drive the mucosa and submucosa through the muscularis speak of increased intraluminal tension. Edel[10] suggests chronic constipation with fecal distention of the large bowel as a source of this increased tension. Beer[11] believes that, in addition to constipation and chronic distention of the large bowel, it is necessary to have the muscular degeneration and weakness which are coincident with old age. Wilkie[6] states that the obstruction of the lumen of one of his appendixes by a carcinoma was sufficient to explain the occurrence of a diverticulum distal to it.

When one attempts to analyze these hypotheses and apply them to appendix diverticula, it is found that, while each one of them may be applicable to a few cases, none of them has a universal application. Klebs' suggestion that the vascular defects in the muscularis are

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7. Villar: Sur un cas d'appendicite chronique avec pseudo-myxoma diverticulaire, Bull. de l'Acad. de méd. **79**:175, 1819.

8. Klebs: Path. Anat., 1869, p. 271.

9. Graser: Ueber multiple falsche Darmdivertikel in der Flexura sigmoidea, München. med. Wchnscher. **46**:721, 1899.

10. Edel: Ueber erworbene Darmdivertikel, Virchows Arch. f. path. Anat. **138**:347, 1894.

11. Beer, E.: Am. J. M. Sc. **128**:135 (July) 1904.

increased in diameter by accumulations of fat about the vessels obviously loses its force when the diverticula occur, as they usually do, in people who are not obese. Graser's hypothesis that chronic distention of the blood vessels in the vascular defects tends to shove aside the muscle bundles, thus increasing the diameter of the defects, does not apply to any of the appendixes with diverticula that I have seen or read about. In these, where there have been broad defects, they have been filled with dense connective tissue, surrounding several rather small and thick walled vessels, which were not distended. It might be argued that these had collapsed after removal, but a majority of the appendixes which we receive for examination in the laboratory reach it with their vessels distended; so this is probably not a valid objection. In any event, this hypothesis has no bearing on the inflammatory defects. There is no proof that chronic constipation and fecal distention per se cause increased intraluminal tension, and it can be definitely stated that diverticula of the appendix at least may occur in people in whom there is no clinical evidence of chronic constipation or fecal accumulation in the large bowel. Beer's suggestion that weakening of the muscle, due to old age, is an etiologic factor is rarely applicable to the appendix diverticula, most of which occur in vigorous adults, and examination will show that the muscular coats are, in almost every case, above the average in thickness. It is probably true that there is some obstruction to the lumen proximally in a majority of appendixes with diverticula; but there are just as certainly others in which there is no demonstrable obstruction, and, therefore, this cannot be regarded as an etiologic factor of universal application.

The importance of increased hydrostatic tension within the lumen of the intestine as a factor in the etiology of diverticula of the intestine (and, by inference, of the appendix) received strong support through the publication, in the latter part of the nineteenth century, of some experimental work that has since been completely discredited, but the influence of which can still be traced in recent articles on the subject. Heschl,[12] Hansemann[13] and Grassberger[14] distended segments of the large intestine with water, and asserted that they saw bulgings along the mesenteric border, which disappeared when the tension was released. They asserted that these occurred at the defects in the mesenteric border through which the vessels pass, and they argued from this that these were weak points through which diverticula might occur in the living intestine, if the increased intraluminal tension were chronic. Beer[11] was

12. Heschl: Wien. med. Wchnschr. **30**:1, 1880.

13. Hansemann: Ueber die Entstehung falscher Darmdivertikel, Virchows Arch. f. path. Anat. **144**, 1896.

14. Grassberger: Ein Fall von multiplex Divertikelbildung, Wien. med. Wchnschr. **10**:149, 1897.

unable to reproduce these bulgings experimentally, and it is probable from Chlumsky's observations that they are at best a phenomenon of dead intestine only. Heschl,[12] Hanau[15] and Good,[16] using human necropsy material, several hours after death, found that, after extreme distention, the intestine burst regularly between the leaves of the mesentery. This observation has been used as an argument to support the hypothesis that the mesenteric border is the weakest portion of the intestine wall, and that perforations and diverticula are therefore more liable to occur in this region than in any other. Chlumsky,[17] however, showed the fallacy of using dead material for investigation by repeating this experiment, using the living dog's intestine within the body. He found that it burst regularly along the antimesenteric border. When he used dead intestine, removed from the body, he found that it burst between the leaves of the mesentery. We are thus left with inconclusive evidence, so far as these experiments are concerned, when we attempt to apply them to determine the forces which tend to produce diverticula in the appendix and the intestine.

The only observations which, it seems to me, can altogether survive critical analysis are the morphologic ones that diverticula are always associated with defects in the muscularis and that the only defects which have been observed are those due to perforation, to scarring following suppurative inflammation and the normal ones in the mesenteric and antimesenteric borders through which vessels pass. There is no proof that diverticula occur through these defects; but all the evidence that we have favors the assumption that they do, and, therefore, it seems fair to accept it as a working hypothesis.

If we turn now to the seven appendixes described in this paper, a reference to Figures 1, 2, 3, 5 and 6 demonstrates very clearly a condition which has not been described before in the literature, possibly because in most of the cases reported the specimens were sectioned transversely instead of longitudinally. In these diverticula, it will be seen that, not only the mucosa and submucosa nearest the muscular defect have passed through it to form an extra muscular sac but also the mucosa and submucosa from the side opposite the muscular defect have passed through it, thus, in effect, diverting the entire lumen outside the appendix into the diverticulum sac.[18] This condition was noted in four diverticula in four different appendixes out of a total of eighteen

15. Hanau: Falsche Darmdivertikel, Virchows Arch. f. path. Anat. **138,** 1894; ibid. **145**:172, 1896.

16. Good: Aus dem Leichenhause des Cantonspitals zu Gallen (Dr. Hanau), Inaug. Diss., Zurich, 1894.

17. Chlumsky: Ueber verschiedene Methoden der Darmvereinigung, Beitr. z. klin. Chir. **25**:539, 1899.

18. Several of the diverticula illustrated by MacCarty and McGrath (Footnote 2) seem to show this condition, but no reference is made to the phenomenon in the text.

diverticula in seven appendixes. The same diversion of the lumen outside the muscularis can be seen in the appendix which had ruptured two years before it was removed, eversion of the mucosa into an abscess cavity resulting, with the proximal and distal portions of the appendix opening into it separately through the muscular defect (Fig. 13).

All of the hypotheses which have been advanced to account for the motive force which pushes the mucosa through the muscular defects depend on a distention of the lumen. The existence of these four diverticula, however, forces us to seek some additional explanation to account for them. It is inconceivable, for me at least, to imagine that the force exerted by the expansion of a fluid or a gas within the lumen of an appendix could *alone* be sufficient to force the entire circumference of the mucosa and the submucosa through a defect in the muscularis. Some other force must be operative in these cases and probably in all the rest of the cases of diverticulum formation as well.

A little reflection will make it apparent that most of the comments and reasoning up to this point have been based on morphologic and mechanical considerations almost exclusively. We have talked of diverticulum formation as if it took place by the action of a force in an inert, lifeless tube made up of tissues of different tensile strength but without any other characteristics. This neglect of the biologic factor has resulted in the neglect of the tremendous energy transformations which are occurring constantly in the appendix and intestine throughout life; it has allowed constant morphologic references to the smooth muscle tissue of the appendix but scarcely any physiologic ones.[19] In other words, the force exerted by the contraction of the smooth muscle tissue in the appendix during life has not yet been considered as an important factor in the etiology of diverticulum formation.[20]

MORPHOLOGIC AND EXPERIMENTAL EVIDENCE IN FAVOR OF THE MUSCULAR CONTRACTION HYPOTHESIS OF DIVERTICULUM FORMATION

Inasmuch as no one, as far as I can find out, has investigated the possibilities of this force in connection with appendiceal diverticula, it

19. Seelig (Footnote 26) in a single phrase suggests that the contraction of the smooth muscle plays some part in diverticulum formation in the appendix, but does not develop the theme.

20. It is all the more astonishing that this should be so when one finds that it has been considered in reference to diverticula elsewhere in the body. Forty-five years ago, Zenker (quoted by Judd: Esophageal Diverticula, Arch. Surg. **1**:38 [July] 1920) described the pharyngo-esophageal dimple just behind the cricoid cartilage on the posterior wall, where there is a weakened point due to the arrangement of the musculature at the junction of the upper esophagus and the lower end of the pharynx. He noted that diverticula occur through this, and believed that the increased pressure exerted on the weak point during the act of swallowing was the chief etiologic factor.

seemed as if it were worth attempting. With this purpose it was determined to state a theorem in regard to diverticulum formation and then to see whether it would serve to explain all the known phenomena. If it did so this would not prove it true, because we know by observation only a few of the phases of the process. If supported by the facts, however, it would make it at least a more satisfactory hypothesis than any which has been advanced before, because, as has been indicated, none of them serve to explain all of the known phenomena.

It has been assumed, therefore, that diverticula take place through defects in the muscular coats, and that the chief motive force that drives the submucosa and mucosa through these defects is that exerted by active contraction of the longitudinal and circular muscles on the submucosa, mucosa and contents of the lumen. With this in mind, a morphologic examination of a number of appendixes has been made and certain experiments have been performed, with the hope that the results would suggest whether the hypothesis was credible or not.

In order to understand the process of diverticulum formation, it is necessary to emphasize certain well known mechanical and physiologic facts. One must first recall the physical relationship which the coats of the appendix bear to one another. The mucosa and the submocosa, with the muscularis mucosae running between, form a single coat, the three parts of which are intimately bound together. The muscularis, subserosa and serosa likewise form a more or less homogeneous whole. But, in many appendixes, especially those which have not been previously badly damaged by inflammation, the attachment between the submucosa and muscularis is by loose bonds of areolar and elastic tissue, so that the two coats are capable of a certain amount of excursion, one upon the other.

The muscular coats of the appendix are divided into an outer longitudinal coat and an inner circular coat, the latter usually much thicker than the former. Under normal conditions of tonus, unless widely distended by inspissated feces, fluid or a fecolith, the mucosa and submucosa in many appendixes are maintained within the circular muscle in a plicated state, both transversely and, to a less extent, longitudinally. When there is a loose attachment of the submucosa to the muscularis, it allows free play between these two coats. With strong muscular contractions, the tendency is to exaggerate these folds by reducing both the diameter and the length of the appendix. If an appendix or intestine is fixed in a state of strong muscular contraction and then sectioned longitudinally, these exaggerated folds can easily be seen.

With these observations in mind, let us suppose that there is a weak point in the muscular wall of the appendix and the muscle contracts: what may happen? In order to answer this question, we are forced to use animals, because it is improper to use the living human appendix in

situ for experimental purposes. While dogs' appendixes are too much like exaggerated cecal pouches to be altogether satisfactory for experimental purposes, I was forced to use them because I was unable to procure organs more nearly resembling the human. Reference to Experiments 1 to 5 will show that, if a muscular defect is made in the wall of a dog's appendix by incising down to the submucosa or by excising a piece of subserosa and muscularis, there is, following the injury, a strong contraction of both circular and longitudinal muscles, and, at the same time, the mucosa and submucosa protrude through the muscular defect, forming a diverticulum.[21] This can persist for some days. If the same thing is done to an appendix sufficiently long after death for the muscular tissue to lose its contractility, no such protrusion will occur, *provided the appendix or intestine is not distended.* If it is distended, the protrusion may occur (Experiment 5).

From these experiments, we see that, with a suddenly produced unfilled gap in the muscularis of a dog's appendix or intestine, because of the free play between the submucosa and the muscularis, the mucosa and submucosa may be protruded by muscular action and may also be protruded by the force of fluid tension acting from within the lumen. We can go a step farther in studying the effects of muscular contraction and distention. If an appendix with a protrusion of mucosa and submucosa through an experimentally produced gap in the muscularis due to muscular contraction is distended moderately, the diverticulum fills with fluid and stands out more clearly as a smooth rounded hemisphere. If the internal hydrostatic tension is considerably increased, the diameter of the appendix gradually increases, the coats of the appendix become thinner as they are stretched, the gap in the muscularis increases in width and the *diverticulum grows steadily less until it disappears entirely* (Experiment 4). This effect is produced apparently because the increased internal hydrostatic tension overcomes the force of muscular contraction and stretches and thins the muscular coats until they are forced outward to as great a distance as the original diverticulum. In a dog's dead intestine, in the body, increased intraluminal

21. When this procedure was repeated, using a human appendix which had just been removed from the body and which showed very moderate chronic inflammatory changes, the same bulging of the mucosa and submucosa through the muscular defect occurred. In this case, the bulging was only 1 mm. beyond the serosal level (Experiment 6). Some years ago, when Dr. Lewisohn (Clinical and Experimental Studies on Congenital Pyloric Stenosis, Surg., Gynec. & Obst. **26**:320 [March] 1918) was doing some experimental Rammstedt operations on the dog's pylorus, in one case, the operation was followed by diverticulum formation. It seems probable that, in this situation, it was due more to muscular contraction than to distention, as the pylorus is probably very rarely distended (compare illustration in his article).

tension at first forces the mucosa and submucosa out through the gap in the muscularis, making a diverticulum; with marked increase in the tension, the appendix dilates enormously, and the diverticulum disappears as described above (Experiment 5).

We are now in a position to state that a diverticulum may occur through an experimental defect in the muscularis of a dog's appendix, both as the result of intraluminal distention and as a result of muscular contraction. Increased muscular contraction increases the tendency for the mucosa to be protruded, while greatly increased intraluminal tension tends to diminish and, eventually, to cause the disappearance of the diverticulum.

With these suggestive animal experiments in mind, let us turn back to the human appendix to see whether a morphologic consideration of a number of appendixes will support the hypothesis that muscular contraction is the chief factor in the causation of diverticula of the appendix and, at the same time, strengthen the view that in many cases the vascular defects are the weak points in the muscularis through which the protrusions occur.

The arteries and veins which supply the appendix reach that organ at from six to eleven different points along the mesenteric border. They send branches that pass through the muscular coats and form a rich anastomotic network in the submucosa. Other branches pass circumferentially in the subserosa, and, at the antimesenteric border, these also pierce the muscular coats and join the submucosal anastomosis. A few small twigs pass into the muscularis between the mesenteric and antimesenteric borders; but they are apparently for the supply of the muscularis itself and do not pierce it. The vessels as they pass through the muscular coats are surrounded by a greater or less amount of connective tissue and sometimes by a few fat cells. When seen, these vascular defects are always larger and much more prominent on the mesenteric than on the antimesenteric border. Usually, the vessels pass through at right angles to the long axis of the appendix; less often, they pass through tangentially. When found in the appendixes examined, they varied between 0.5 and 1 mm. in thickness on the mesenteric border, and were always less than 0.5 mm. on the antimesenteric border.

In examining the appendixes with diverticula in the series reported in this paper, it was noted in all of the cases that the muscular coats appeared above the average in thickness, while in all but one the vascular defects were very prominent and easily visible.[22] An attempt was made to determine the relationship between the width of the vascular defect

22. In the one exception, the diverticulum was believed to have followed an inflammatory weakening of the wall rather than to have occurred through a vascular defect (Case 7).

and the thickness of the musculature in the appendix. Ninety-six appendixes were chosen at random as they were received in the laboratory of surgical pathology, and the thickness of the muscular coat was measured by a millimeter scale. The average thickness was 0.86 mm., a figure slightly above the 0.5 to 0.75 mm. which Aschoff [23] says is the variation in normal adult appendixes. During the routine gross examination of these appendixes, it has been a laboratory rule that the appendix shall be bisected longitudinally in a plane passing through the mesenteric and antimesenteric borders, and the presence or absence of the vascular defects noted in the report. When the defects are of any size, the vessels are surrounded by dense connective tissue, which shows up as a dull white band, in sharp contrast to the much darker semitranslucent muscularis. It is not unreasonable to assume, therefore, that, in those cases in which the defects were seen, they were probably broader and contained more connective tissue than in those in which they were not seen. In the forty-two cases in which they were seen, the average thickness of the muscularis was 1.06 mm. (the thinnest, 0.5, and the thickest, 3.0 mm.); while in the fifty-four cases in which the vascular defects were not seen, the average thickness of the muscularis was 0.71 mm. (the thinnest, 0.3, and the thickest, 1.5 mm.).[24] The average diameter of the vascular defects in the mesenteric border in the forty-two cases in which they were seen was 0.5 mm.

Turning to the series of seven appendixes with diverticula, we find the average width of the muscularis 1.91 mm. (the thinnest, 1, and the thickest, 2.7 mm.). In Cases 1 to 6, inclusive, in which the mesenteric vascular defects were noted passing through the muscularis, the average diameter of the largest defects noted in each case was 1 mm. (the largest, 1.5, and the smallest, 0.5 mm.).

These figures suggest that appendixes vary considerably in the thickness of their muscular coats and that the broader vascular defects which are more easily visible with the naked eye are found in the more thickly muscled appendixes. They also indicate that the muscularis of those appendixes in which diverticula are formed averages a whole millimeter thicker than in the average appendix removed at operation, and that the mesenteric vascular defects in the appendixes with diverticula tend to be broader than the average.

We cannot satisfactorily account for this muscular thickening in every case by the hypothesis that it is hypertrophy resulting secondarily

23. Aschoff, L.: Die Wurmfortsatzentzündung, Jena, 1908, pp. 10 and 72.

24. Of course, these figures have only comparative value because of the many variables and sources of error, the greatest being the edema of the muscularis in acute cases. But, as the same factors of error are operative in both series, it is felt that the value of the results for comparison is not vitiated.

from obstruction of the lumen and increased function of the muscular tissue, this increased function being stimulated by accumulation of contents within the lumen, because, while in five of the seven cases there was either actual or potential obstruction of the lumen proximal to the diverticula, in two (Cases 4 and 6), there was neither, and both of these had thick muscular coats. So, while admitting the attractions of this hypothesis and suggesting with Wilkie [6] that it plays some part, it must be admitted that some other factor or factors are involved. In any event, one cannot escape from the suggestion that these thick muscles indicate increased function and that they are capable of more powerful contraction, and hence of exerting a more powerful force, than the thinner ones.

It will not be unprofitable, at this point, to reconsider the question of diverticulum formation as a result of damage to the wall of the appendix from suppurative inflammation. As we have seen, it has been stated, first, that an intramural abscess may form, destroy the muscularis, drain into the lumen of the appendix, and subsequently be replaced by scar tissue, thus leaving a defect through which it is conceivable that a diverticulum may pass; second, that, during an acute inflammatory attack, the appendix may perforate, and, as a result of the perforation, a diverticulum or mucocele may develop. All that can be adduced to support the first statement are the observations that intramural abscesses occur; that scars occur in the muscularis, and that diverticula occur. All of the intervening steps are suppositious. In regard to the second, however, we are able to demonstrate more of the intervening steps, and it will be worth while to do this because of the light which it seems to throw on the dynamics of the process.

It must be recalled that the vast majority of perforations of the appendix are associated with extensive necrosis of the mucous membrane. Most perforations appear as holes lined with necrotic tissue, leading through the wall from a lumen also largely lined with necrotic tissue. Occasionally, however, we find the condition of affairs illustrated in Figure 12, in which there is a wide perforation without extensive necrosis, which has resulted in the protrusion and eversion of the mucosa and submucosa (Case 8). A condition analogous to this, but of two years' standing, can be seen in Figure 13, which has been referred to before. There we find perforation, with protrusion through the perforation not only of the adjacent mucosa but also of the entire lumen, so that the proximal and distal portions of the lumen open separately into the extra-appendiceal abscess (Case 9).

Figure 4 presents a tract lined with mucous membrane leading to a cavity in the mesenteriolum near the tip, filled with mucus and pus and lined with granulation tissue. As there is a diverticulum in another part of this appendix, this may be thought of either as a diverticulum in

which the acute inflammatory process has destroyed its lining mucosa or (and this appeals to me as more probable) it may be considered that, as the result of an acute inflammatory process in the appendix wall, possibly during a previous attack, a perforation and partial protrusion of the mucosa occurred with the formation of a mucocele, and that the inflammation present when the appendix was removed involved a previously formed sac.

The speed and ease with which mucosal defects in the intestine can regenerate is well known, so that it requires no great stretch of the imagination to surmise that, in Cases 8 and 9, had there been a subsidence of the infection, perhaps by free and adequate drainage of the abscess through the fistulous tract leading back through the wall of the appendix into the lumen and so on into the cecum, a regeneration of mucosa might have lined the extramuscular sacs, giving the picture seen in other diverticula in this paper; in the latter case with a single opening into the lumen, in the former with a double opening. Figure 11 illustrates a diverticulum near the tip of an appendix that may very well have occurred in this way.

Figure 12 illustrating a fresh perforation, with protrusion of the mucosa and eversion, cannot fail to recall, to all who have seen it, the behavior of the mucosa of any part of the living gastro-intestinal tract when the entire thickness of the wall is cut through and the lumen entered. It will be remembered that, coincidentally with the wide gaping of the wound, the mucosa protrudes and, as it comes out, turns back on itself so that it both projects beyond the surface of the serosa and still allows the cut edge of the mucosa to lie in contact with the cut edge of the serosa. If the same cut is made in a dead intestine, none of these phenomena occurs. In this case, we cannot blame the intraluminal tension; for, as soon as the lumen is entered, the intraluminal and extraluminal tensions are equalized. We are therefore compelled to fall back on the force of muscular contraction which responds to the irritation incident to the trauma of the incision. The segment cut into is narrowed and shortened by the contraction of the circular and longitudinal fibers, which, in turn, forces the redundant mucosa and submucosa through the opening; and, because the submucosa is attached to the muscularis while the mucosa is a free, unrestrained surface, eversion occurs. The similarity between this experimental end-result and the perforations illustrated in Figures 12 and 13 is so striking that I believe we are justified in adopting the hypothesis that the force resulting from muscular contraction is the chief factor in producing the eversion of the mucosa and submucosa.

SUMMARY

The foregoing reexamination of the process of diverticulum formation in the appendix has set forth some experimental evidence and

some morphologic facts, all of which have tended to support the muscular contraction hypothesis with which we started, and none of which have contradicted it. To recapitulate, it has been shown that there must be a weak point in the wall of the appendix which is probably due either to an especially broad vascular defect in the mesenteric or antimesenteric border filled with connective tissue, a condition found in most appendixes with diverticula; or to a scar in the muscularis, the result of a suppurative process; or to perforation during an acute attack, with protrusion of the mucosa and subsequent subsidence of the infection followed by regeneration of the mucosa within the former extramuscular abscess cavity.

It has been suggested that, in all forms of diverticula without exception, contraction of the circular and longitudinal smooth muscle coats is probably the chief active factor in driving the mucosa and submucosa through the weak point in the muscularis. In support of this, there is the morphologic evidence that, in all seven of the cases reported, the muscularis is above the average in thickness. In five of these, this is associated with more or less obstruction of the lumen proximally either by intraluminal mechanical means, complete obliteration, twists or angulations. In the other two, no obstruction could be demonstrated, but it was possibly present during life. Usually, this obstruction is accompanied by dilatation of the lumen, distal to it. There is also the condition of complete diversion of the lumen outside the muscularis into the diverticulum sac, which can be explained best by the muscular contraction hypothesis.

By experiment on dogs' appendixes, it has been demonstrated that operative muscular defects result immediately in protrusions of mucosa and submucosa accompanied by active contractions of the circular and longitudinal muscles, and that, when the muscles have lost their contractility, no protrusion will occur, if the lumen is empty. It has also been shown that moderate distention of the lumen will result in a similar protrusion, while extreme distention results in obliteration of the diverticulum.

From the foregoing data, the following attempt has been made to reconstruct the process of diverticulum formation. An appendix in which a diverticulum occurs must be thought of as a muscular tube above the average in thickness, and, because increase in structure connotes increase in function, capable of unusually powerful contraction, both longitudinally and transversely. It always contains submucosa, muscularis mucosae, mucosa and more or less material within the lumen. These different elements are relatively inert and should be thought of as the contents of the muscular tube, even though some of them are loosely attached to its inner surface. If such a muscular tube contracts, it becomes a potentially closed cavity, and there is exerted a

strong pressure on the contents, which may be considered as fluid, and so governed by the laws of hydrostatics. If there is a weak point in the muscular tube incapable of withstanding this transmitted force, it must yield, and the contents will protrude through the gap to a point where the driving and resisting forces become equalized. Depending on the balance between these two forces, the protruded elements may consist only of mucosa and submucosa immediately adjacent to the defect, or they may consist of the entire contents of the muscular tube in the segment containing the defect. In the former case, there will be a diverticulum consisting of a simple pouch lined with mucous membrane; in the latter, complete diversion of the lumen into the diverticulum sac.

REPORT OF CASES

APPENDIXES WITH DIVERTICULA

CASE 1.—July 3, 1922, a specimen was removed at necropsy by Dr. B. M. Vance from a woman, aged 46, whose history was unknown (medical examiner's case).

Gross Examination.—The specimens were the cecum, ascending colon, terminal ileum and appendix. The appendix was about 6 cm. long and sinuously curved. Near its base a band extended from the parietal peritoneum to the mesentery of the appendix. Almost opposite this, another broad band extended from the terminal ileum and its mesentery to the appendix and its mesentery. These were probably congenital. The appendix varied in diameter from 5 mm., near the base, to 10 mm., just beyond the middle. When the cecum was distended, apparently no fluid passed into the appendix. The distal half of the appendix had a free mesentery, containing a considerable amount of fat. In this could be seen, buried in fat, two hemispheric bulgings, which were apparently continuous with the mesenteric border of the appendix.

A longitudinal section revealed that the lumen in the proximal part was patent, but measured only about 1 mm. in diameter and contained a small amount of thick, slimy greenish material. At a distance of 3.7 cm. from the cecal-appendiceal junction, the lumen came to an abrupt termination after being diverted to one side. A thin septum separated it from the terminal portion of the appendix, where the lumen was widely dilated up to 5 mm. and filled with thick, white flocculent material. In this portion of the appendix, there were two defects in the muscularis of the mesenteric border, through the more proximal of which a protrusion of mucosa and submucosa had occurred for a distance of 4 mm. Through the more distal gap, not only had the mucosa and submucosa of the same side protruded, but also all the rest of the mucosa and submucosa of that region, so that the entire lumen had come to lie outside the appendix, within the sac of the diverticulum. From the diverticulum, the lumen passed back within the surrounding muscular coats once more in its course toward the tip, becoming markedly constricted as it did so. At the tip itself, there was a third defect in the muscularis through which the mucosa and submucosa passed, dilating up to 3 mm. to form a terminal diverticulum, which was surrounded by the mesenteric fat at the tip. The muscular coat of the appendix throughout was thick, averaging from 1.5 to 2 mm. The vascular defects in the mesenteric border were for the most part slender white lines about 0.3 mm. thick; one, however, measured 1 mm. (Fig. 1).

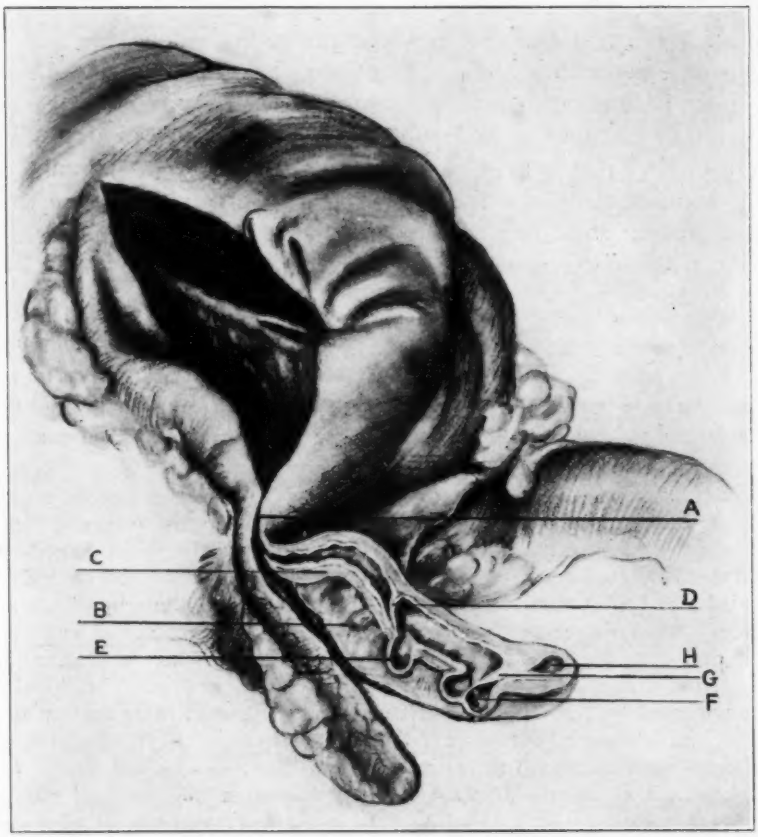

Fig. 1 (Case 1).—Appearance of necropsy specimen from a woman, aged 46, with unknown history. The appendix has been bisected longitudinally. *A* is the junction of the appendix with the cecum; *B* the mesenteriolum. The muscular coat *C* is easily distinguished, by its darker appearance, from the white appearing mucosa and submucosa. At *D*, a septum of mucosa and submucosa narrows the lumen and diverts it toward the mesenteric border, so that the proximal portion of the lumen is completely separated from the distal. Beyond this, the lumen is widely dilated, and the mucosa and submucosa pass through a defect in the mesenteric border forming the simple pouchlike diverticulum *E*. At *F* is another diverticulum with complete diversion of the lumen outside the muscular coats. The septum of submucosa and mucosa *G* appears complete in the plane of the section, but there was a small hole in it through which the continuity was maintained, indicated by the black marker in the diverticulum. *H* is a diverticulum at the tip.

Microscopic Examination.—Sections from the terminal diverticulum revealed that the sac of submucosa and mucosa projected for only a short distance through the muscular defect. It was intimately adherent to the surrounding fat, and there were many dilated veins immediately about it. The mucous membrane lining the sac was thin and poorly supplied with glands, and the submucosa showed only an occasional small lymph follicle. Sections of the large diverticulum into which the lumen was diverted confirmed the gross findings in every way. The thin septum which passed from the wall of the appendix opposite the muscular defect, through and completely across the diverticulum to its apex, was composed of a central core of submucosa lined on both sides by mucosa. As in the diverticulum, at the tip the mucosa was thin and poorly supplied with glands, and the submucosa contained only an occasional lymph follicle. The subserosa was everywhere thick and well supplied with dilated blood vessels. The muscular coats appeared thick and intact, and were without evidence of previous inflammation.

CASE 2.—*History.*—F. K., man, aged 30, married, a German fireman, suffered from cramps and vomiting for three days. He had had no previous similar attacks, and was never constipated. There was tenderness, and a mass was palpable in the cecal region. The temperature was 99.4 F. The white blood cells numbered 15,400; polymorphonuclears, 83 per cent.

Operation (Dr. Joelson, supervised by Dr. W. B. Parsons).—May 27, 1922: The appendix was acutely inflamed, laterad to the colon, extended up along the right side of the cecum, and was bound down by recent fibrinous adhesions. It was excised from the tip back, and the wound was drained. Recovery followed. June 12, 1922, there was a small granulating area still unhealed in the abdominal wound. Otherwise the patient was well.

Gross Examination.—The specimen was about 12 cm. long. It was slightly angulated at the junction of the proximal and middle thirds. The proximal third was about 9 mm. in diameter. Beyond this, it plunged into an adherent swollen mass of fatty tissue, which was dusky red and covered with a flaky, fibrinous exudate. The diameter of this mass was 2.6 cm. on the average (the appendix itself in this portion measured 1.7 cm. in diameter); 1.2 cm. from the base, there was a rounded, bulbous excrescence, slightly pedunculated and averaging 1 cm. in all diameters, projecting from the mesenteric border. It was soft and reddened and had infiltrated fibrous tissue and fibrin adherent to its dome.

On longitudinal section, the appendix was found to be divided into three sharply defined zones. In the *proximal portion,* the lumen was a narrow slit less than 1 mm. in diameter and apparently empty. It extended in a straight line for a distance of 1.5 cm., until it reached a point opposite the middle of the diverticulum, where it bent sharply at a right angle toward the mesenteric border and, accompanied by the mucosa and submucosa of both the mesenteric and antimesenteric borders, passed through a broad defect in the thick muscularis. Here, it joined, by a mere potential slit, the *second portion* of the appendix. Beyond this slit, the lumen was abruptly dilated, forming the main cavity of the diverticulum. The lumen then passed back through the same defect in the muscularis, where it was somewhat narrowed and, turning again at a right angle, resumed its course along the center of the long axis of the appendix. This portion was 1 cm. from the apex of the diverticulum to the center of the appendix and 2.5 cm. along its long axis. It was filled in this portion with thick pale creamy material under slight tension. The muscularis in this portion had been torn away along the mesenteric border for a short distance, probably by operative trauma. The second portion ended abruptly as

a blind pouch, the lumen at this point being completely separated from the distal portion. The *third portion* comprised a dilated lumen filled with bloody pus and lined with necrotic, hemorrhagic granulation tissue. There were two broad defects passing through the muscularis at the mesenteric border which seemed to be composed of reddened, congested fibrous tissue. The broadest was 1.2 mm. One similar, but much more slender, fibrous thread passed through the muscularis of the antimesenteric border. Surrounding the distal half was the tremendously thickened edematous fat of the adherent mesentery. On making further sections through the tip longitudinally, another diverticulum was revealed, with a neck passing through the muscularis about 1 mm. broad, beyond which was a cavity 3 mm. in diameter in the surrounding mesenteric fat. At its thickest part, the muscularis measured 1.5 mm. in width. The width of the broadest vascular defect was 1.2 mm. (Fig. 2).

Microscopic Examination.—The section through the large diverticulum, near the base, revealed that it was lined with flattened mucous membrane and that the thick, creamy material filling it was made up almost exclusively of

Fig. 2 (Case 2).—Appearance of an acutely inflamed appendix from a man, 30 years old. There were no previous attacks. At *A* is shown a diverticulum in the mesenteric border near the base, with complete diversion of the lumen of the appendix into the diverticulum, because of the projection of the tongue of mucosa and submucosa *B* through the muscular defect (compare Fig. 3). At *C*, the lumen is completely interrupted by a septum of connective tissue, beyond which there is partial necrosis of the mucosa coincident with the acute inflammation. *D* and *D* are two vascular defects filled with connective tissue in the muscularis of the mesenteric border; *D'* is a similar defect in the muscularis of the antimesenteric border; *E*, region from which Figure 4 has been made.

leukocytes. The dilated central portion of the appendix which was continuous with this diverticulum was lined with a mucosa of similar type. The mucosa of the proximal portion was more normal in appearance. The neck of submucosa which extended from the antimesenteric side of the appendix through into the diverticulum was composed only of fibrous and fatty tissue, which was covered by the muscularis mucosae and mucous membrane. The diverticulum was covered exclusively by submucosa and muscularis mucosae, outside of which was adherent fatty tissue. A vessel of considerable size passed down

alongside the neck; which suggested that the diverticulum occurred in one of the normal vascular defects in the muscularis of the mesenteric border (Fig. 3).

Sections from the tip of the appendix revealed that the mucosa was extensively destroyed by a suppurative process. Where it was preserved, it was represented by a single layer of columnar cells, with only a few short mucous glands scattered at intervals between them. At a point opposite the abscess,

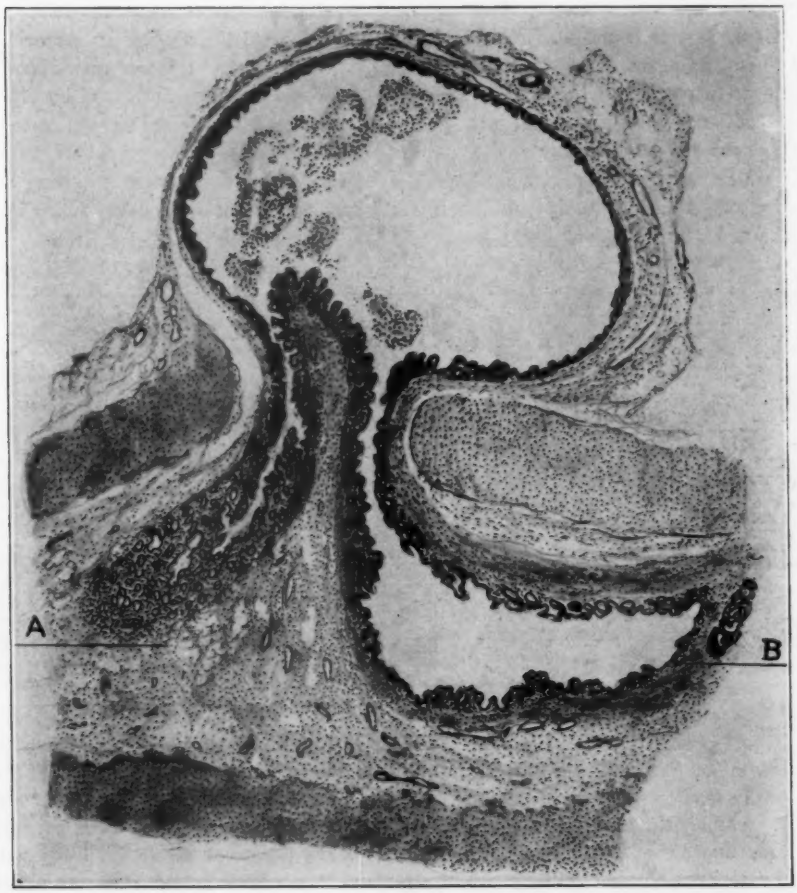

Fig. 3 (Case 2).—Appearance of microscopic section of diverticulum *A* shown in Figure 2: diversion of lumen surrounded by mucosa and submucosa through defect in muscular coats. The section is through a plane which shows the afferent and efferent sections of the lumen passing through the vascular defect, but does not show its continuity toward the cecum at *A*, nor toward the tip at *B*. This continuity is apparent in Figure 2.

there was a defect in the muscularis, and through this, mucous membrane extended to a point almost opposite the external coat. Here it ceased and the rest of the cavity was lined with granulation tissue and filled with a mass of leukocytes mixed with mucus and mononuclear cells (Fig. 4).

The condition in the tip suggested that a mucocele was probably in the course of formation in the distal isolated portion with a diverticulum at its tip. An infection involved the tip, causing a perforation of the diverticulum, which had resulted in abscess formation in the mesentery. The central portion had probably been involved secondarily in the acute inflammatory reaction, causing an empyema involving this portion and its extension into the proximal diverticulum. The small proximal portion attached to the base had practically escaped involvement at this stage of the infection.

Case 3.— *History.*—L. W., a colored woman, aged 20, single, an American, who was doing maid service, had suffered pain in the right lower quadrant for the past two weeks, which had grown steadily worse and was increased by urination. There was no nausea, but she had vomited twice. Two similar attacks had occurred, one and two and one-half years ago, with fever which lasted from four to six weeks. In the past, she had four attacks of pain, swelling and redness of the left ankle and shoulder. She was usually constipated. Promiscuous intercourse was admitted.

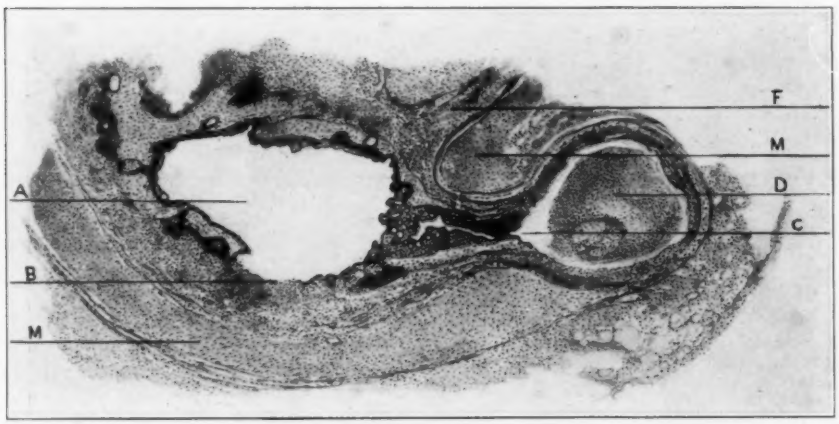

Fig. 4 (Case 2).—Drawing of microscopic slide of section made through tip of appendix at *E* in Figure 2, but at a deeper plane. The lumen of the appendix is shown at *A*, surrounded by inflamed mucosa which has been destroyed at *B*. At *C* is the opening of a mucous membrane lined tract which leads through a defect in the muscular coats *MM*, into an extra muscular cavity *D*, lined with granulation tissue and filled with mucus and pus. At *F* is a connective tissue filled vascular defect passing from the mesenteriolum through the muscular coats to the submucosa.

Examination.—There was marked tenderness low down in the right lower quadrant and slight tenderness in the left lower quadrant. The cervix was large, soft and irregular. There was intense pain when the cervix was moved. There was a tender mass in the right fornix and slight tenderness in the left fornix. The patient was observed in the hospital for eleven days, during which time she improved greatly. The temperature ranged from 101.4 to 98 F. The white blood cells numbered 8,100; polymorphonuclears were 81 per cent.; hemoglobin, 70; the red blood cells numbered 4,600,000. The Wassermann reaction was negative.

Operation (Dr. Joelson, supervised by Dr. J. Greenough).—March 13, 1922: Right salpingo-oophorectomy, left salpingectomy, myomectomy of small uterine fibromyoma and appendectomy were performed. The appendix was retrocecal and entirely retroperitoneal. Uneventful recovery ensued. May 13, 1922, the wounds were fully healed, and the patient felt relieved of symptoms.

Gross Examination.—The specimen was an appendix 6 cm. long and varying from 5 to 8 mm. in diameter. The surface was dull and roughened. The superficial vessels were injected. There were several small firm white nodules on the mesenteric and antimesenteric borders, averaging about 2 mm. in diameter. On the mesenteric border near the tip, there was a much larger one, projecting 4 mm. from the surface and measuring 5 mm. in diameter. On longitudinal section, the lumen of the appendix was found to be patent to within 7 mm. of the tip; from 3 to 4 mm. in diameter, and filled with inspissated fecal material. The wall of the appendix averaged 2 mm. in thickness, of which the muscularis occupied 1 mm. One fibrous band was seen passing through the muscularis in the mesenteric border, which was 0.8 mm. in diameter. The terminal 7 mm. of the appendix seemed to show first an obliteration of the mucosa and its replacement by fibrous tissue; beyond this the lumen was a mere potential space surrounded by a small amount of mucous membrane. It seemed to pass out into the large nodule projecting from the mesenteric border. (A more detailed description of this appendix cannot be given for it was unfortunately mislaid after this preliminary examination had been made). With the appendix were a fibromyoma of the uterus, two chronically inflamed tubes and a cystic ovary.

Microscopic Examination.—Sections from the tip of the appendix revealed a diverticulum formed by the extension of the submucosa and mucosa through a defect in the muscular layers. On one side of the diverticulum, a slender strand of the inner circular muscle had been carried through the gap with the submucosa and extended almost to the highest portion of the dome of the diverticulum. There were two distinct lumina in the diverticulum, lying side by side as it passed through the muscularis, each lined with mucosa and submucosa, which join in the extramuscular sac. A good sized vein lay alongside the neck of the diverticulum as it passed through the muscularis. A section taken from one of the smaller nodules along the mesenteric border described in the gross presented a mass composed of connective tissue surrounding a number of relatively large thick walled veins passing through a gap in the muscularis and widening out to form a thicker rounded mass outside the muscularis, in this way simulating a diverticulum, although no mucous membrane appeared in it. (This is an example of the false diverticulum described by Seelig[25]). None of the sections presented any signs of acute inflammation.

Case 4.—*History.*—E. W., man, aged 54, a widower, American, had suffered for thirty-six hours from generalized pain in the abdomen and vomiting. He had had no previous similar attacks but had had vague gastric disturbances and cramps occasionally in the past. There were tenderness and muscular spasm about the umbilicus and in the mesial portions of both lower quadrants. The temperature was 100.6 F. The white blood cells numbered 21,000; the polymorphonuclears were 93 per cent.

Operation (Dr. Greenough).—Jan. 27, 1922: Appendectomy with drainage was carried out. The appendix lay behind the ileocolic junction in a region

25. Seelig: False Diverticula of the Vermiform Appendix, Ann. Surg. **44**: 78, 1906.

of localized peritonitis. A stormy postoperative course was attended by a pulmonary embolus with infarction of the lung. June 6, 1922, there was bulging of the abdominal wound, but the condition was otherwise good.

Gross Description.—The specimen was 4 cm. in length, 1.3 cm. in diameter near the base and 1 cm. in diameter at the tip. It was reddened and edematous throughout, but more markedly so in the distal half, which was covered with a greenish-yellow fibrinous exudate. The mesenteriolum was also swollen and edematous, with a broad attachment. It was covered with a fibrinous exudate throughout its length. On longitudinal section, it could be seen that the thickening in the proximal half was due to a tremendous thickening of the submucosa. The muscular coat was 2 mm. thick. Along the mesenteric border, there were eight vascular defects from 1 to 1.5 mm. in thickness, passing through it at right angles to the lumen. On the antimesenteric border, the vascular defects were barely visible. The lumen for a distance of 2.3 cm. was small but patent and passed with slight deviations nearly through the center of the appendix; then it made a right angled turn and, accompanied by the mucosa and submucosa, passed through the muscular coat to form a dilated sac

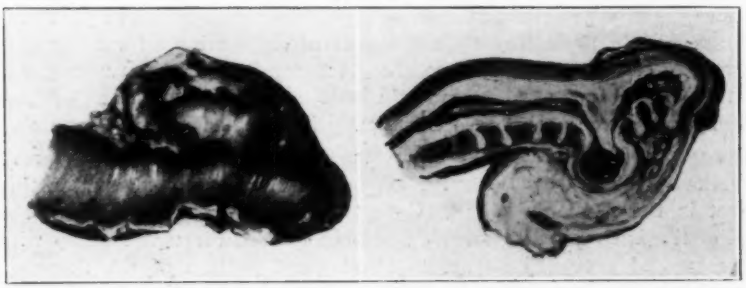

Fig. 5 (Case 4).—Appearance of acutely inflammed appendix from a man of 54, who had had no previous attacks. At the left is the external appearance of the appendix, with the thickened mass of the mesentery above, with fibrin adherent to it. To the right is a longitudinal section showing the single diverticulum in the mesenteric border with complete diversion of the appendix lumen into it. The thick muscular coats appear as a broad dark band, interrupted along the mesenteric border by the diverticulum and by seven sharply contrasted connective tissue filled vascular defects.

5 mm. in diameter between the leaves of the mesenteriolum. It then proceeded directly back toward the center of the appendix, paralleling its former course outward, and passing through the same muscular defect. Having reached the center of the appendix, it proceeded on its course to the tip. The muscular defect through which the two arms of the lumen passed measured 4.5 mm. in width. We thus had a complete diversion of the lumen through the muscularis into the mesenteriolum. At one point, the mucosa lining the diverticulum was defective, indicating a pin point perforation into the mesentery (Fig. 5).

Microscopic Examination.—Longitudinal sections revealed that the diverticulum in the mesentery had a lumen 5 mm. in diameter, lined with intact mucosa surrounded by submucosa and the thickened edematous, inflamed mesentery. Only one of the mucosal tubes connecting it with the site of the original appendix lumen was shown in these slides. This was also lined with

intact mucosa as it passed through the broad defect in the muscularis. Several other broad defects in the muscularis were seen, through which passed blood and lymphatic vessels and dense connective tissue. Surprisingly little evidence of the acute inflammatory reaction appeared in the section except in the form of a fibrinopurulent exudate on the serosa and fibroblastic reaction in the mesentery, together with a moderate edema and dilation of lymphatic vessels (Fig. 6).

CASE 5.—*History.*—J. F. D., man, aged 21, single, American, a clerk, suffered sudden sharp cramplike pains in the abdomen which were not relieved by any measures taken. The bowels moved, and he vomited once. He had had six similar attacks during the past year. He was usually constipated. The tempera-

Fig. 6 (Case 4).—Photomicrograph of the appendix shown in Figure 5. The diverticulum lined with mucosa and submucosa in the mesenteric fat is shown at *A*. The afferent portion of the lumen leading from the cecal end of the appendix to the diverticulum sac is shown at *B*. The continuity of the diverticulum with the lumen of the appendix at the tip *C* is not shown in this section but is clear in Figure 5. *DDD* are four of the broad defects in the mesenteric border of the muscularis filled with blood vessels and connective tissue. The ball-like expansions of these outside the muscular coats (so-called false diverticula) are well shown.

ture was 100.6 F. The white blood cells numbered 17,600; polymorphonuclears, 90 per cent. Tenderness was noted on both sides of the midline below the umbilicus. There was rectal tenderness anteriorly, and marked distention of the colon.

Operation (Dr. H. A. Murray, supervised by Dr. W. G. Penfield).—Jan. 9, 1922: The appendix was retrocecal, apparently perforated near the tip, swollen, reddened, and covered with fibrin. A quantity of free, gray purulent

fluid was found in the peritoneum, which grew *B. acidilactici.* Appendectomy was performed, with drainage. The wound healed slowly, but convalescence was otherwise smooth.

July 18, 1922: The scar had a slight tendency to bulge; otherwise the patient was well.

Gross Examination.—The specimen was an appendix about 5 cm. long and 0.8 cm. in diameter. It was slightly curved, reddened and edematous, and the distal half was dusky red and covered with a thick, flaky fibrinous exudate. The mesentery was thickened and attached along the convex border. No diverticula were visible externally, nor was there external evidence of perforation.

On longitudinal section of the organ, it was apparent that at the base for a distance of 5 mm. the lumen of the appendix was completely occluded and replaced by fibrous tissue. Distal to this the lumen was widely dilated and lined with a smooth layer of mucous membrane. It contained cloudy fluid. It was at once apparent that there were three complete diverticula along the mesenteric border in the distal half. Two of these measured about 4 mm. in diameter and consisted apparently of protrusions of mucosa and submucosa

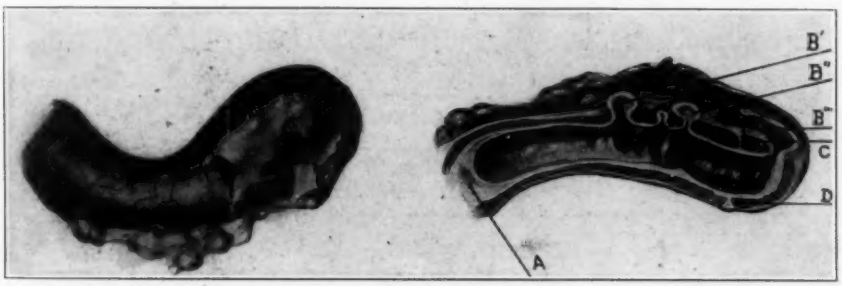

Fig. 7 (Case 5).—Appearance of the acutely inflamed appendix of a man, aged 21, who had had six preceding acute attacks. To the left is the external appearance, showing the tip covered with fibrin and the mesenteriolum attached along the convex border; to the right a longitudinal section, showing complete obliteration of the lumen at the base, *A,* beyond which it is widely dilated. *B'*, *B"* and *B"'* are three diverticula in the mesenteric border. *C* is a muscular defect in the mesenteric border, and *D*, a muscular defect in the antimesenteric border. Between *B'* and *B"* is what appears to be an incomplete diverticulum. This is incorrectly drawn, as it is a defect similar to *D.*

through defects in the muscularis, which were about 2 mm. in diameter. The most distal diverticulum near the tip was much larger when it reached the mesentery, spreading out over an area nearly 1 cm. in length. It was filled with semigelatinous, mucoid material and there was a perforation through one end into the mesenteric fat. It was noticeable that there were, in addition to the complete diverticula, two defects in the muscularis along the mesenteric border and one along the antimesenteric border, all of which were filled with dense, white connective tissue. The defect on the antimesenteric border was capped with a small ball of dense white tissue in the subserosa. No lumen or mucosa appeared to be present in these three areas. The muscularis measured about 2 mm. in thickness. The broadest point was 2.2 mm. in thickness. The connective tissue filled defects averaged 0.5 mm. in diameter (Fig. 7).

Microscopic Examination.—A section taken near the tip revealed that the largest diverticulum was a protrusion of submucosa, muscularis mucosae and mucosa through a large defect in the muscularis, into the tissue between the leaves of the mesentery. It had spread out considerably in this situation and at its proximal end had a defect in its lining mucous membrane which communicated directly with a mass of mucus lying free in the tissues. This mucus was mixed with round cells and leukocytes. In the surrounding tissues, there was edema, congestion of vessels and cellular infiltration. On the serous surface was adherent an exudate of fibrin and leukocytes (Fig. 8). The other two diverticula were of the usual type. There were also defects in the mesenteric border of the muscularis, filled with connective tissue which surrounded thick walled veins. The lack of signs of inflammatory reaction in the appendix itself made one suspect that the infection started possibly in the mucocele which came from the diverticulum at the tip of the appendix.

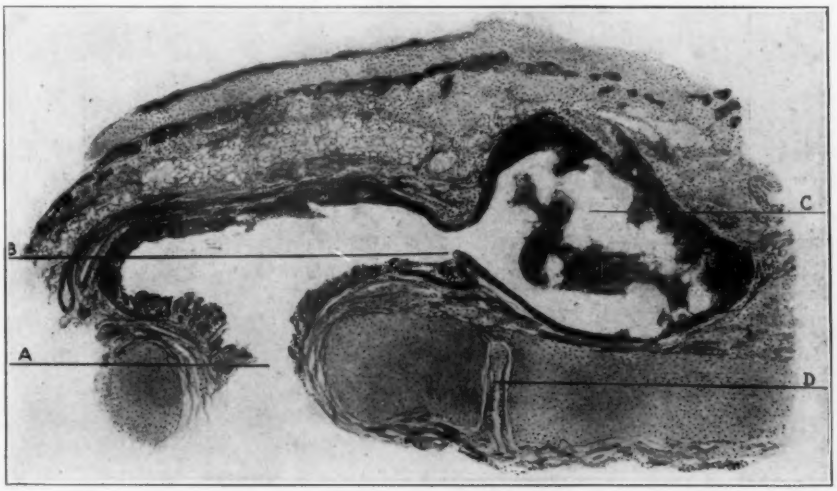

Fig. 8 (Case 5).—Drawing made from the microscopic section of diverticulum *B'''*, Figure 7. At *A* is shown the neck of the diverticulum leading to the extra muscular cavity which is lined with mucous membrane except at *B*, where it stops at the small opening shown in the illustration. This leads into an accessory cavity *C*, which is lined with the connective tissue of the mesentery and contains mucus and leukocytes. The mucous membrane lining the lumen of the appendix was torn away in making the section. Here, we have a diverticulum with an infected mucocele leading off from it. *D* is a vascular defect.

Case 6.—*History.*—M. F. C., woman, aged 28, single, American, a bookkeeper, came to the hospital to have the appendix removed because of a recent acute attack of appendicitis.

Examination.—On admission, tenderness was elicited on deep palpation in the right lower quadrant. All other signs were negative. The temperature was 99 F. The white blood cells numbered 7,100; polymorphonuclears, 66 per cent.

Operation (Dr. F. B. St. John).—July 23, 1921: Appendectomy was performed. The appendix was found in the pelvis adherent to the surrounding

structures by recently formed soft adhesions. Three nodules were noted along the antimesenteric border and one at the tip. The patient made a good recovery.

Gross Examination.—The specimen measured 6.5 cm. in length and 1 cm. in diameter in the distal half. A large mass of mesenteric fat was attached distally. It was pink and smooth, except in a few scattered areas from which adhesions had been torn. At the tip was a firm rounded pale mass, 7 mm. in diameter. Three similar rounded projections, smaller in size, were seen on the antimesenteric border in the distal half. On longitudinal section, it was seen that the appendix was somewhat irregularly curved, with its concavity toward the mesentery. The lumen appeared to be patent throughout, varying from 2 to 3 mm. in diameter. White lines representing vascular defects filled with connective tissue could be seen in both the mesenteric and the antimesenteric

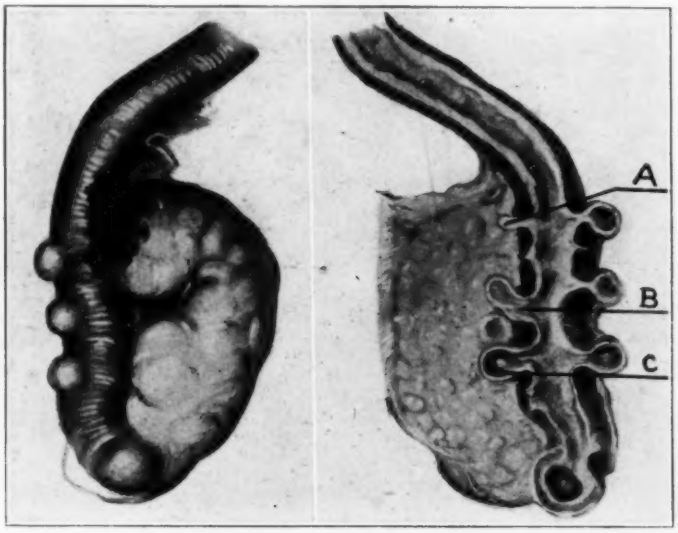

Fig. 9 (Case 6).—Appearance of appendix removed from a woman of 28. in an interval following an acute attack. To the left is the external appearance of the appendix showing the three nodules on the antimesenteric border, one at the tip and the large mass of fat in the mesenteriolum; to the right, a longitudinal section showing these four diverticula and three others in the mesenteric border, at *A*, *B* and *C*. It will be noted that the lumen is patent throughout and remains within the muscular coats, although diverticulum *B* has apparently two cavities. (For an explanation of this, compare the gross description of Case 6.)

borders. The broadest one was in the former, measuring 1 mm. in width. It was at once apparent that there were seven diverticula in the appendix: three in the mesenteric and three in the antimesenteric border, and one at the tip, which was the largest. All of these seemed to be simple protrusions of the mucosa and submucosa through the muscularis, which averaged 2.7 mm. in thickness, except the middle one on the mesenteric side, which was a more complicated affair. There seemed to be a fold or septum of mucosa and submucosa in the neck of the diverticulum as it passed through the muscularis, so that in one half of the cut section it appeared as if there were two lumina;

while in the opposite half there appeared to be only one. Outside the muscularis, the diverticulum appeared to have two distinct mucosa lined cavities (Fig. 9). This seemed to be an intermediate stage between the usual simple unilateral protrusion of the mucous membrane and the complete deviation of the lumen into the diverticulum sac, illustrated in the first four cases.

Microscopic Examination.—Longitudinal sections of the appendix taken through the middle revealed three of the diverticula, two on the mesenteric and one on the antimesenteric border. It revealed the tremendously thick mus-

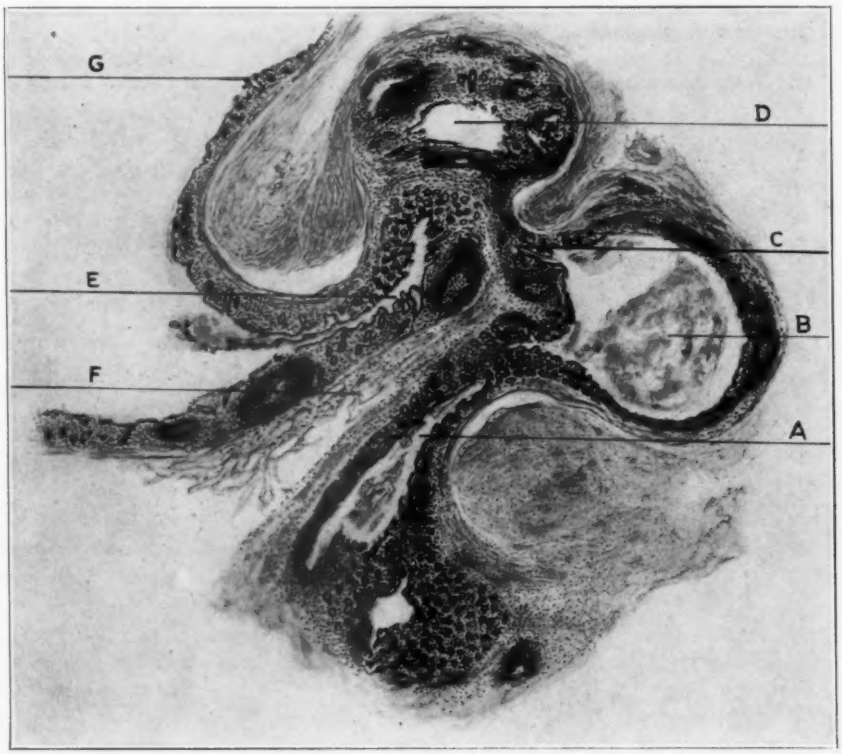

Fig. 10 (Case 6).—Appearance of microscopic section of diverticulum, *B*, shown in Figure 9. The section was made from the half of the appendix opposite that illustrated in Figure 9. In this illustration, the lumen, *A*, passes through the muscularis from the proximal portion of the appendix to an extramuscular cavity, *B*. This, in turn, communicates with a second adjacent cavity, *D* via *C*, and the lumen then passes back within the muscular coats at *E*. *G* indicates a portion of diverticulum *C*, Figure 9. Although the tongue of submucosa, *F*, apparently occludes the lumen, this condition obtains only in this half of the appendix and not in the opposite half, illustrated in Figure 9.

cular coat, which had a minimum amount of connective tissue in it. Two of the diverticula were simple protrusions of mucosa and submucosa through muscular defects. In the other one, the lumen of the appendix appeared to be completely diverted, passing through the muscular defect into two mucosa lined cavities in the mesentery, which were connected by a narrow passage;

and then passing back again through the muscular defect. These bulbous enlargements were filled with cellular and amorphous débris. In the surrounding fat, there were a few round cells and leukocytes. There was no evidence of acute inflammation (Fig. 10). In places where vascular defects without diverticula passed through the mucularis, the density of the connective tissue surrounding the veins and lymphatics was demonstrated. In one instance, a rounded mass of this dense connective tissue with a mass of tortuous vessels included in it could be seen capping the muscular defect. This reached a diameter of 4 mm. and was an example of the false diverticulum described by Seelig.[25] The serosa was thickened and edematous, and presented many dilated capillaries and lymphatics.

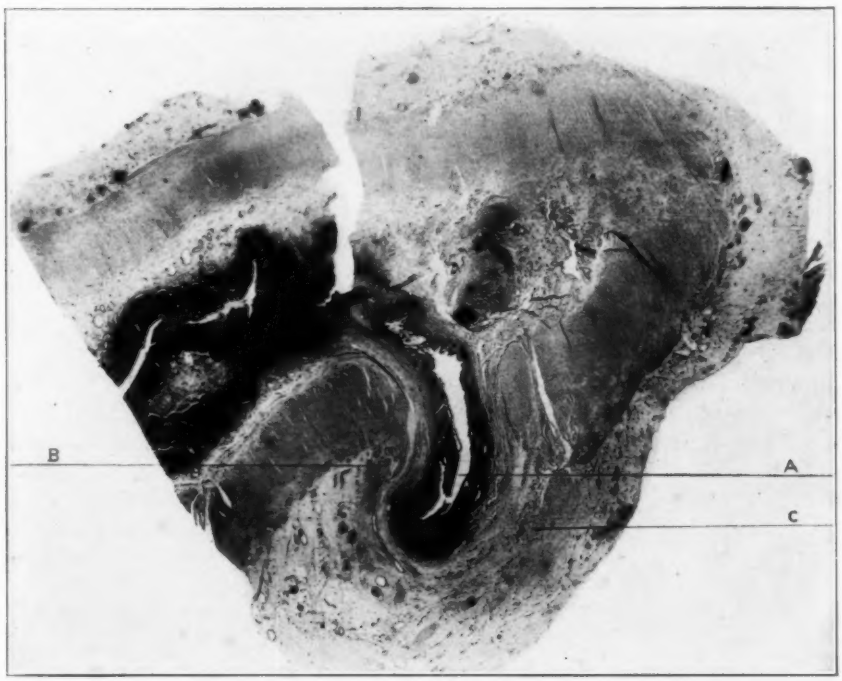

Fig. 11 (Case 7).—Photomicrograph of appendix from a man of 20, removed during an acute attack, with a history of four or five preceding attacks. The section shows the tip of the appendix with a diverticulum, *A*, lined with mucosa passing through a defect in the muscularis. At *B* and *C* can be seen fragmented smooth muscle bundles, which have apparently been carried out for a short distance with the protrusion of the mucosa.

Case 7.—*History.*—J. R., man, aged 20, single, American, a farmer, had had pain and soreness in the right lower quadrant for two days. The bowels had not moved for two days. Four or five mild attacks of similar nature had occurred in the past six months. The patient was not usually constipated. There was tenderness in the right abdomen, most marked in the right lower quadrant, with rigidity. There was rectal tenderness high on the right. The temperature was 102 F. The white blood cells numbered 16,000; polymorphonuclears, 85 per cent.

Operation (Dr. Jameson).—April 18, 1915: Appendectomy with drainage. was performed. The cecum was undescended and of the infantile type. The appendix was retrocecal, and an abscess surrounded the tip, which was up under the liver. There was cloudy fluid in the pelvis. The patient made a good recovery. When last seen, twenty-one months after operation, he was perfectly well.

Gross Examination.—(As the specimen has been lost, the following is a copy of the examination made in 1915.) The specimen was 8 cm. in length. It was sharply angulated at the junction of the proximal 3 cm. with the distal 5 cm. The proximal portion was 1 cm. in diameter and, after making the right angled turn, it disappeared into a mass of edematous infiltrated fat. In the distal portion, the wall was edematous and infiltrated. The mucosa appeared gangrenous, the lumen was patent but was filled with purulent and fecal material; 1 cm. from the tip, there was a diverticulum of the lumen into the surrounding fat. It projected for a distance of 5 mm. and was somewhat bulbous, the broadest part measuring 5 mm. in diameter. The muscularis averaged about 2 mm. in thickness. As the appendix had not apparently been cut in a plane passing through the mesenteric and antimesenteric borders, no vascular defects could be seen.

Microscopic Examination.— Section made through the tip of the appendix revealed that the mucous membrane in this portion was intact and, with the muscularis mucosae and submucosa, passed out through a defect in the muscularis for a short distance, into the surrounding fat. All the coats in this region were edematous and were infiltrated with leukocytes and round cells. This was most marked in the extramuscular fat, where, in addition, all the vessels were engorged and there had been considerable proliferation of fibroblasts. It was interesting to note that a few bundles from both the longitudinal and the circular muscle coats had been carried outward along the side of the diverticulum nearly to its apex (Fig. 11). Aschoff [25] quotes von Brunn [26] to the effect that this phenomenon is seen in connection with diverticula which have followed inflammatory damage, and serves to distinguish them from the other type. The evidence is all in favor of this being an example of a diverticulum resulting from a former perforation of the appendix and protrusion of the mucosa, with subsequent repair and regeneration of the mucosa within the extramuscular tissue.

PERFORATED APPENDIXES

Case 8.—*History.*—J. Z., man, aged 31, married, Hungarian, a janitor, had an acute attack of abdominal pain, with constipation, lasting two days, finally localized in the right lower quadrant. There had been no previous attacks. The patient had never been constipated before. There were marked pain and tenderness in the right lower quadrant and right flank. Rectal examination was negative. The temperature was 101.8 F. The white blood cells numbered 22,000; polymorphonuclears, 92 per cent.

Operation (Dr. F. B. St. John).—Jan. 16, 1916: Appendectomy was performed with drainage. The appendix was retrocecal and had ruptured. There was a localized abscess about it, which contained a concretion. Culture of pus showed *B. faecalis alcaligenes* and streptococcus. Two years after operation the patient was perfectly well.

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26. Von Brunn: Ueber Divertikelbildung bei Appendicitis, Beitr. z. klin. Chir. **46**:67, 1905.

Gross Examination.—The specimen was 4 cm. long. The proximal half was swollen, reddened, and covered with a fibrinous exudate. In the antimesenteric border, there was an area of perforation nearly 1 cm. in length. The mucosa in this area appeared to be everted and was covered with a thick, greenish yellow exudate. The distal half of the appendix was swollen and reddened, but was not nearly so much affected as the proximal part. With the appendix was a fecal concretion which was rounded and firm, and measured about 1 cm. in diameter.

Fig. 12 (Case 8).—Photomicrograph of a cross section through a perforation of an acutely inflamed appendix. The broad defect involving one third of the circumference can be plainly seen, and at *A* and *A′* the extent of the eversion of the protruded mucous membrane, which at *A′* is practically continuous with the serosa.

Microscopic Examination.—The section was made transversely through the perforation. All of the coats were much distended with edema and massively infiltrated with leukocytes. The defect in the lumen was in the antimesenteric border and occupied about one third of its circumference. The muscularis had retracted so that it occupied only about one half of the circumference of the appendix. The mucosa and submucosa projected out beyond the muscularis and were everted so that on one side the mucosa reached to the serosa, while

on the other side it extended to a point opposite the middle of the muscularis. The mucosa within the appendix was relatively intact, but the protruded portion was denuded of surface epithelium and was represented by edematous granulation tissue, in which the tubular glands appeared. There was no lymphoid tissue in any part of the submucosa (Fig. 12).

Case 9.—*History.*—S. A., woman, aged 23, single, Spanish, a dressmaker, had an attack beginning about thirty hours before operation, of nausea, vomiting and generalized abdominal pain, which later became localized in the right lower quadrant. Two years before she had had an attack of pain in the right lower quadrant, accompanied by fever and vomiting which lasted eight days and during which a mass could be felt in the right lower quadrant. She was treated with morphin and ice bags.

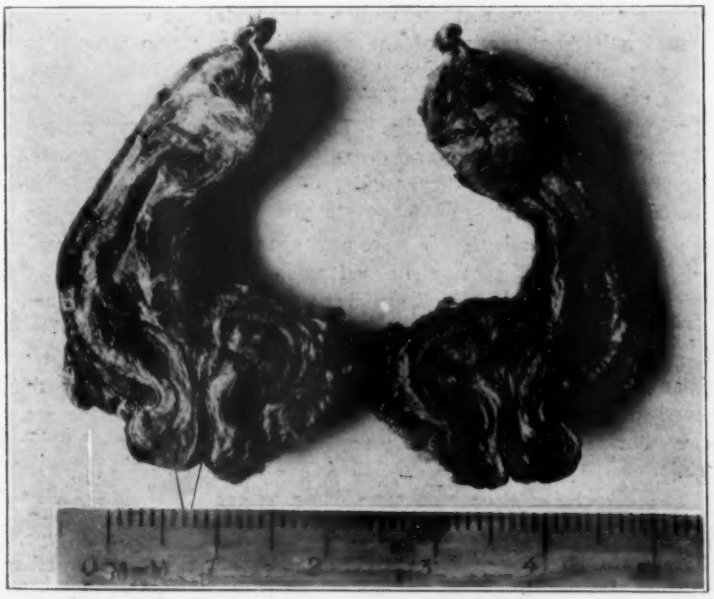

Fig. 13 (Case 9).—Appearance of appendix removed from a woman of 23 during an acute attack, with one previous acute attack two years before. The appendix has been divided longitudinally. The appendix has perforated, with protrusion of the entire lumen. The two wire markers have been passed into the separated portions. The entire nodular area surrounding these two wires on the external surface of the appendix is covered with mucous membrane.

Examination.—On examination, at the Presbyterian Hospital, there were rigidity and tenderness over the lower right rectus, extending out into the flank. No masses were felt. The temperature was 99 F. The white blood cells numbered 12,000; polymorphonuclears, 81 per cent.

Operation (Dr. J. Greenough).—Aug. 8, 1921: Appendectomy was performed, with drainage. The appendix was buried in dense adhesions and was incorporated in the wall of the cecum, starting posteriorly and curving forward along the lateral aspect. There was an old perforation near the tip, from which the mucosa pouted. No holes could be found in the cecal wall. Pus escaped from

the appendix during removal. Following operation, the patient had a secondary hemorrhage in the wound, suppurating parotid sialadenitis, bed sores on the back and heels, and cecal fistula. Jan. 17, 1922, the cecal fistula was closed by operation. March 22, incision and drainage of a subhepatic abscess was carried out. June 10, 1922, all wounds had healed; the patient felt well and was gaining weight.

Gross Examination.—The specimen was 5.5 cm. long and markedly curved throughout its length. It was swollen, reddened, and covered with the shaggy remnants of recently separated adhesions. Near the tip in the antimesenteric border was a rounded, elevated area covered with reddened mucous membrane about 1.3 cm. in diameter and raised 5 mm. above the surface of the appendix. In this, there were two pin point openings, one of which was the mouth of a tract leading toward the tip of the appendix, while the other was the external opening of a tract communicating with the lumen of the appendix toward the base. On longitudinal section of the appendix, these two mucous membrane lined tracts could be seen passing through a defect in the muscularis 6 mm. wide. Thus, there was a complete diversion of the lumen of the appendix into the extra-appendiceal abscess cavity. The muscularis near the tip measured about 1.5 mm. in thickness. Several broad white bands in the mesenteric border of the appendix represented the vascular defects. The broadest of these was 1 mm. wide (Fig. 13).

Microscopic Examination.—We were not successful in getting a section which included both tracts in the same slide. However, they showed the mucosa and submucosa passing outside of and well beyond the muscularis and then turning outward and downward until it met the serous coat at a considerable distance from the point at which it passed through the muscular defect. It was well formed, and, in its extra-appendiceal portion, the cells making up the glands were mostly of the goblet variety. The coats of the appendix were somewhat edematous, but presented only a very moderate infiltration with leukocytes and round cells.

PROTOCOLS OF EXPERIMENTS

Experiment 1.—A young adult female mongrel of the hound type, weight about 25 pounds (11.4 kg.), was operated on, Oct. 27, 1921, under ether anesthesia, the appendix and cecum being delivered into the upper part of a right rectus incision. The appendix had a separate mesentery and, except for the tip, was bound down closely to the cecum. It emptied easily into the cecum and refilled almost immediately as soon as the pressure was removed. An area 6 by 8 mm. near the tip was denuded of serosa, subserosa and muscularis by sharp dissection with the knife. At once, the submucosa and mucosa bulged through the defect, forming a dome shaped swelling 4 mm. in height. At the same time, it was noted that the appendix contracted both in diameter and in length, and its wall became very hard. After some seconds, this muscular spasm was relaxed, and, coincidently with the softening of the appendix wall and the increase in its dimensions, the diverticulum appeared slightly less prominent and its walls slightly less distended. It never disappeared. A ligature, together with an attached piece of omentum, was now tied about the middle of the appendix, passing through a hole in the mesenteriolum; this constricted the appendix considerably, and it was hoped that, with the formation of scar tissue, a permanent obstruction would be obtained. The abdominal wound was then closed.

The dog, which had been suffering before from distemper, died of that disease, Nov. 18, 1921, twenty-two days after the operation. At necropsy, a

moderate number of adhesions of omentum to the appendix were found. The terminal ileum, cecum, ascending colon and appendix were removed from the body en masse and distended with liquor formaldehydi (formalin) before being studied.

Gross Examination.—The diverticulum was now about 8 mm. in diameter and passed through a muscular defect 5 mm. in diameter. It was, therefore, slightly larger than before. Where the omentum and silk ligature were tied

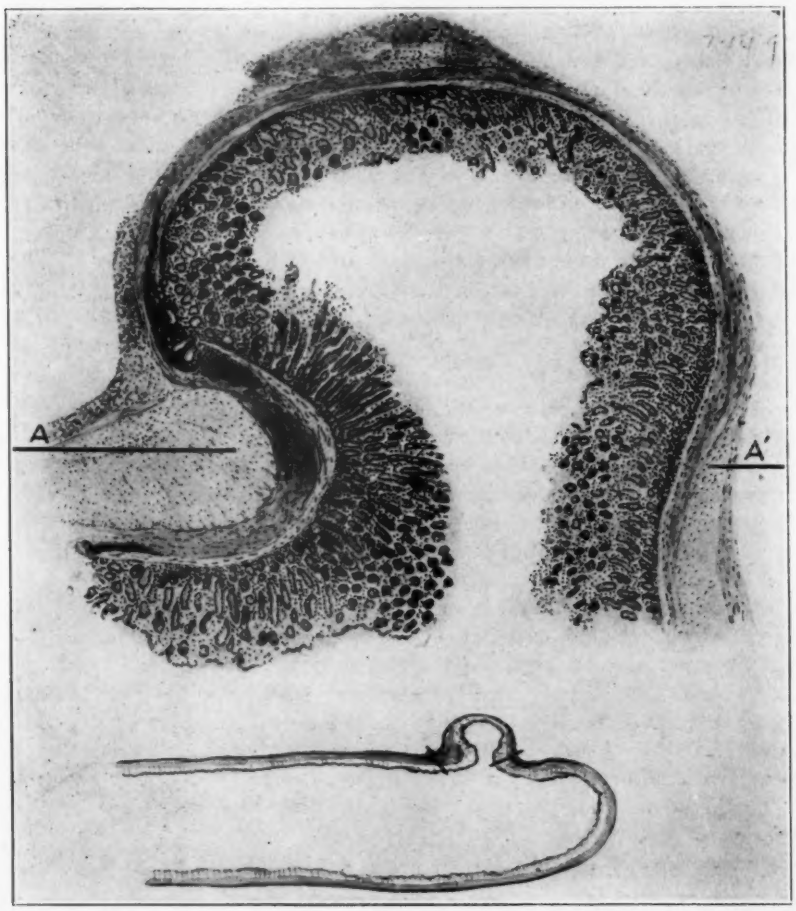

Fig. 14 (Experiment 1).—Appearance of distal portion of dog's appendix showing experimentally produced diverticulum twenty-two days after the muscular defect was made. Above is a drawing made from a microscopic section of the area included between the heavy lines in the sketch. It shows at *A* and *A'* the ends of muscle coats with the protruding mucosa and submucosa passing out to form an extra muscular pouch. Attached to the apex of the diverticulum is a torn adhesion.

about the appendix, the lumen was decreased to one half in its diameter, but there was no obstruction.

Microscopic Examination.—A section through this diverticulum was identical with that of "acquired" diverticula in the human appendix. It showed a

protrusion of mucosa, muscularis mucosae and submucosa through a muscular defect. Attached to the exposed submucosa was some adherent omentum (Fig. 14).

Experiment 2.—A small male mongrel was operated on Dec. 30, 1921, under ether anesthesia, the technic being a repetition of that employed in Experiment 1. In this case, two defects were made: one, 5 mm. square, by excision of muscular coats, the other, by a simple incision 1 cm. long down to the submucosa. In this case, there was a bulging of the submucosa and mucosa through the defects when the appendix contracted, but it was not marked and almost disappeared with relaxation. A thick doubled silk ligature was tied very tightly about the middle of the appendix, so that the lumen was obstructed.

The dog underwent another experiment performed on his spleen during the same operation, and died two hours later, with shock and mesenteric thrombosis. At necropsy, the appendix was found covered with fibrinous exudate and a small amount of blood. It was soft and flaccid, and, although there was a slight bulge of mucosa and submucosa through the defect, there was no true protrusion forming a sac as in Experiment 1.

Microscopic Examination.—Section showed that the muscularis had been completely removed and that the mucosa bulged through to the level of the surrounding serosa.

Experiment 3.—A young adult female mongrel terrier was operated on, March 20, 1922, under ether anesthesia, the appendix and cecum being mobilized and the appendiceal-cecal junction carefully freed, care being taken to injure as few as possible of the large vessels in the mesentery. After crushing with clamps, the base of the appendix was divided; phenol was applied to the stump, and it was inverted into the cecum and buried by suture. The distal divided end was first opened, so that a small piece of cork about 8 mm. in diameter could be inserted into it; and then phenol was applied, and it was inverted and buried by suture. There was some soiling by appendiceal contents during this stage of the operation. A defect 5 mm. square was made in the wall of the appendix over the cork, exposing the submucosa. At once, the appendix contracted, and the mucosa and submucosa protruded through the defect, forming a small dome shaped swelling about 2 mm. high. This persisted. The wound was then closed. This operation produced a completely segregated appendix with a foreign body in it and a diverticulum.

The dog developed distemper, and died April 10, twenty-one days after operation.

Necropsy (April 11, 1922, twenty-seven hours after death.).—There was a suppurative tracheitis, bronchitis and beginning bronchopneumonia. Considerable postmortem discoloration was noted in the peritoneal cavity. The region of the appendix was completely covered with firmly adherent omentum. When this was separated, the appendix was found lying just as at operation. The diverticulum was well defined and appeared as before. The cork was now in the lumen, but slightly nearer the tip. There was no distention of the segregated appendix. The intestines were empty except for a minimal amount of fecal material in the large intestine. The duodenum, with the pancreas in its mesentery, had become intimately adherent to the site of operation. Fluid could be passed only with difficulty through the cecum. After distention with liquor formaldehydi, the whole area was removed for study. When the specimens were opened, it was found that the appendix had perforated and there was a closed

abscess cavity outside the base, the walls of which were formed by the cecum, duodenum and pancreas. The cork lay just beyond the site of the diverticulum. The lumen of the appendix was not dilated, averaging 3 to 5 mm. in diameter. It contained some cloudy fluid mixed with débris. The diverticulum measured 1 cm. where it passed through the muscularis, and it projected for a distance of 3 mm. above the surrounding serous surface. The cecum, where the wall had been inverted, was sharply angulated and its lumen reduced to a semilunar slit, 6 mm. from horn to horn and 0.5 mm. at its broadest point. There was fecal material, however, on both sides of this narrowed area.

Microscopic Examination.—Sections of the diverticulum revealed a bulging of submucosa, muscularis mucosae and mucosa through a defect in the muscularis. None of the muscularis remained over the denuded submucosal surface. Sections made through the point of perforation of the appendix and the abscess cavity revealed that the walls of the latter were made up of adherent duodenum and cecum, both of which were relatively normal, except that the serosal surfaces were covered with a necrotic layer where they bordered the abscess cavity, and there was a portion of pancreas which presented extensive degenerative and fibrotic changes. The abscess cavity contained many mononuclear cells and a few leukocytes, together with much amorphous débris.

Experiment 4.—A dog which was about to be killed was selected and anesthetised with ether. A defect 4 mm. square was made in the appendix near its tip, down to the submucosa. The submucosa and mucosa at once protruded from 2 to 3 mm. through the defect, and at the same time the strong muscular contractions which involved the entire organ made it hard, shorter and of smaller diameter. It was observed for some minutes, during which time the muscular contractions were relaxed and then it contracted once more. During the period of relaxation, the diverticulum seemed to become somewhat flaccid, although its total projection was not less.

A metal syringe was now passed through a slit in the cecum up into the appendix and a silk ligature tied about the appendix base over the nozzle, in an attempt to make the appendix a closed cavity. Water was then injected. At first, the lumen of the appendix filled with water, which passed into the diverticulum, and it stood out as a firm, smooth hemisphere above the surrounding serosa. As more and more water was injected, the diameter of the appendix continued to grow greater, and as it did so, the projection of the diverticulum above the surrounding serosa grew steadily less, until there was no longer any projection at all, although the muscular defect had practically doubled in diameter. With all the force which could be exerted through the syringe, an attempt was made to burst the appendix, but this did not occur, as the increased tension caused the ligature tied about the base of the appendix to cut through so that it leaked at this point. The attempt was repeated several times, with the same result. The dog was then killed.

Experiment 5.—A dog was killed with chloroform, and the peritoneal cavity was opened and left exposed to the air. From time to time, bits of serosa and muscularis were excised from the wall of both the large and the small intestine. Each time, there was the usual muscular contraction, resulting in the narrowing and shortening of the segment injured and a protusion of the submucosa and mucosa through the defect. As time passed, the force of the muscular contraction grew more feeble until, about four hours after the death of the animal, there was no longer any response to the injury. The extent of the protrusion through the gap was variable, and seemed to be influenced by the

Summary of Cases of Diverticula of Appendix

Case	Sex *	Age	Previous Attacks of Acute Appendicitis	Habitually Constipated	Removed During Acute Attack	Diverticulum Involved in Acute Inflammation	Duration of Acute Symptoms	Position of Appendix	Lumen of Appendix Obstructed Between Diverticulum and Cecum		Diameter of Diverticula, Mm.	Diverticula					Vessels Alongside Neck of Diverticulum	Width of Broadest Vascular Defect in Mesenteric Border of Muscularis, Mm.	Vascular Defects Seen in Antimesenteric Border	Greatest Thickness of Muscularis, Mm.	Whole Lumen Diverted	Associated Condition
									By Angulation or Twisting	By Obliteration of Lumen and Mucosa		Number	Along Mesenteric Border	Along Antimesenteric Border	At Tip	Elsewhere						
1	♀	46	?	?	At necropsy	.....	.....	Pelvic brim	Yes	No	From 3 to 4	3	2	0	1	0	Yes	1	No	2	1	
2	♂	30	No	No	Yes	In one	3 days	Laterocolic	Probably	In one	From 3 to 5	2	1	0	1	0	Yes	1.2	Yes	1.5	1	
3	♀ (colored)	20	Probably not	Yes	No	.....	.....	Retrocecal and retroperitoneal	Possibly (inspissated feces in it)	Yes	5	1	1	0	0	0	Yes	0.8	No	1	1	Salpingitis, cystic ovary, fibromyoma
4	♂	54	No	?	Yes	Yes	1½ days	Behind ileocecal junction	No	No	5	1	1	0	0	0	Yes	1.5	Yes	2	1	
5	♂	21	6 in 1 year	Yes	Yes	In one	10 hours	Infracecal	No	Yes	From 4 to 10	3	3	0	0	0	Yes	0.5	Yes	2.2	No	
6	♀	28	1	?	No	.....	.....	Pelvic	No	No	From 3 to 5	7	3	3	1	0	No	1.0	Yes	2.7	No	
7	♂	20	4 or 5	Yes	Yes	No	2 days	Retrocecal	Yes	No	5	1	0	0	0	1	No	?	?	2	No	

* In this column ♀ indicates female, ♂ male.

amount of the contents of the lumen. When a loop was emptied of its contents, however, and a defect then made, with the contraction of the muscular tissue there was a protrusion.

When there was no longer any response of the muscular tissue to injury, a defect was made through the muscularis of the appendix, exposing the submucosa; but no protrusion occurred through it. It was possible, by manipulation, to force some of the loose flaccid mucosa and submucosa through the gap, and when manipulation ceased, a fold remained projecting very slightly through the gap. When this procedure was repeated in the intestine, the results varied; if the loop selected was not empty, a protrusion followed the defect; if it was empty, no protrusion occurred.

A syringe was then inserted through the cecum into the appendix and tied into place. When it was distended through this, as in Experiment 4, the first result was the protrusion of the flaccid mucosa and submucosa, forming a smoothly rounded hemisphere above the surrounding serosa. With the tension increased, the sequence of events was exactly the same as in Experiment 4. The diameter and length of the appendix slowly increased, while the projection of the diverticulum grew steadily less, until it ceased entirely. Attempts to burst it succeeded only in causing leaks where the ligature was tied about the base of the appendix.

Experiment 6.—C. D., aged 33, married, American, a housewife, during the previous four months had had three attacks of acute, cramplike pain, starting in the epigastrium, and radiating to the lower quadrant and around both sides to the back, with vomiting. A bowel movement gave relief. The attacks lasted about one day, and tenderness persisted in the right lateral quadrant after attacks. On examination, there was tenderness in the right lower quadrant and right fornix. The white blood cells numbered 10,600; polymorphonuclears, 70 per cent. The temperature was 98.8 F. Aug. 25, 1922, three days after admission, appendectomy was performed by Dr. Freeman, Jr., supervised by Dr. W. B. Parsons. The appendix was retrocecal and twisted on itself. The proximal two thirds was covered by a vascular veil.

This appendix was taken immediately after removal and laid on a cork mat beside a millimeter scale. A defect 6 by 6 mm. was made through the muscularis of the antimesenteric border near the tip. When first laid out, the length of the appendix was 7.2 cm. and the average diameter 6.5 mm. At the end of fifteen minutes, it measured in length 6 cm. and in diameter 7.5 mm. At the end of one hour, it measured in length 5.5 cm. and in diameter 7.5 mm. During the first few minutes, the submucosa and mucosa pouted through the muscular defect until they projected 1 mm. beyond the serosa. They remained in this position without change during the rest of the hour of observation.

The appendix was then distended with fluid through the cut end. It passed freely to the tip and, with the inflow of fluid, the appendix increased in length up to 7 cm., and the projection of submucosa and mucosa through the defect returned within the muscular wall, so that the diverticulum was ironed out and disappeared.

The appendix was then fixed in liquor formaldehydi. On longitudinal section, the muscularis was found to vary from 0.4 to 0.9 mm. in thickness. Microscopic sections revealed some scarring of the inner muscular coat and round cell infiltration of all the coats.

The patient made a good recovery, and the appendix was believed to have been the seat of the trouble.

PRIMARY CARCINOMA OF THE MALE URETHRA*

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Carcinoma of the male urethra is generally considered a rare disease. In 1907 Preiswerk [1] reported two cases and made a careful review of the literature, which at that time contained reports of forty cases, to which he added his two.

Since 1907 isolated case reports have been published, but, as far as I am aware, no extensive review of the literature has appeared. In a recent review, I have been able to find reports of thirty-seven cases; but some of these were published prior to 1907 and evidently were overlooked by Preiswerk. If this number is added to the forty-two cases previously reported, the total is seventy-nine. With the case recently under my observation, which I shall report herewith, the total number at present is eighty. J. W. Thompson Walker's [2] three cases, because of the lack of data, are not included in the foregoing calculation.

Aside from the interest in this disease, due to its rarity, the case herewith reported is interesting because of its pathology. The gross and microscopic pathology was that of a papillary carcinoma, a rare type of urethral carcinoma, since the largest number hitherto reported have been of the pavement or squamous-cell type. In addition to the papillary carcinoma, the entire urethra was studded with benign papillomas.

REPORT OF CASE

History.—Dr. N., aged 53, referred by Dr. Ralph Hissem, was admitted to the Presbyterian Hospital, Feb. 16, 1921, complaining of swelling, induration and tenderness of the shaft of the penis with fistula formation; enlarged inguinal glands; frequency and burning on urination, and urgency and dribbling afterward. He had had the usual diseases of childhood. There was no history of venereal disease. Twenty-five years previously, a large mass of papilloma had been removed from the urethra with a curet.

In November, 1920, the patient had experienced some irritation about the glans. There was a discharge from the urethra, perhaps due to the presence of a very long prepuce. He had had similar trouble several times, because of a long prepuce; and he had consulted a physician who advised argyrol injections, but by mistake he had used Hartzell's solution (iodin crystals, 50 grains; zinc iodid, 15 grains; potassium iodid, 15 grains; water, ½ ounce, and glycerin, ½ ounce). In about an hour, the prepuce became enormously swollen; the next day blood exuded from the meatus, followed several days later by swelling of the penis. Difficulty of urination occurred, and his condition

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* Read before the Chicago Pathological Society, Feb. 12, 1923.

1. Preiswerk: Ztschr. f. Urol. **1**:273, 1907.

2. Walker, J. W. T.: Surgical Diseases and Injuries of the Genito-Urinary Organs, New York, Funk and Wagnalls Company, 1914.

became worse. An abscess formed on the inferior surface of the penis, which was lanced, December 15, resulting in the formation of a urinary fistula which had been present ever since, the opening having increased in size on account of an overgrowth of what was diagnosed at that time as granulation tissue. Numerous small abscesses had formed since, causing multiple fistulas. Acute retention occurred about the middle of December, at which time Dr. Hissem made a dorsal slit in order to expose the meatus. The penis was swollen to the size of the patient's forearm. He had fever, toxemia and extravasation of urine. Sounds up to a No. 31 were passed under ether, and an indwelling catheter was inserted for four days. The bladder was irrigated because of pus, and sounds were passed every two or three days. The large sizes produced bleeding.

It was never noticed that the inguinal glands were enlarged until the last few days before admission. They were never tender. Frequency had been present until a month previously. Burning was present in the urethra during urination. Urgency was severe, the patient being unable to retain the urine at times. Dribbling occurred after urination. There was no hematuria, epididymitis nor backache.

Physical Examination.—The patient presented the appearance of a fairly strong man. He was of slight build, and apparently was not acutely ill. Examination of the heart and lungs was negative. Neither tenderness nor masses were discovered in the abdomen.

Genitalia: Examination of the testicles was negative; the spermatic cords were tender but not enlarged. The inguinal glands on both sides were enlarged and slightly tender. The penis was greatly enlarged, being 7 cm. in diameter and about 20 cm. in length. On the lower surface, about the middle, was a mass of red granulation tissue, 3.5 cm. in diameter. Beside it was a fistula, 1 cm. in diameter. The urine flowed through this mass on urination. The shaft was very hard, not very tender and moderately reddened, and the induration extended down to the symphysis and then stopped. Posteriorly, it could be felt extending to the perineum. The prepuce was very thick. Pus was present at the meatus.

Rectal Examination: The prostate was small and soft, and the seminal vesicles were palpable.

Blood Examination: Leukocytes numbered 10,300; hemoglobin, 69 per cent. Blood pressure was: systolic, 155; diastolic, 84. The Wassermann reaction was negative.

Operation.—Feb. 26, 1921, under ether anesthesia, amputation of the penis, transplantation of the urethra into the perineum and removal of the inguinal glands en masse were carried out. The first step in the operation was the removal of the inguinal glands. The patient made an uneventful recovery.

Histologic Examination.—The pieces removed from the large tumor showed papillary carcinoma.

Inguinal Glands: Six different sections from six different glands revealed no evidence of carcinomatous infiltration. The glands were filled with large numbers of round cells. The diagnosis was chronic adenitis.

PATHOLOGY

Primary carcinoma of the male urethra may be classified as follows: (1) squamous-cell carcinoma; (2) columnar-cell carcinoma; (3) papillary carcinoma, and (4) adenocarcinoma.

The squamous-cell carcinoma is, by far, the most frequent type of carcinoma which occurs in the urethra. In Preiswerk's tabulation of thirty-nine cases, thirty-one belonged to this group. In the thirty-six cases collected in this review, twenty-four were of this type. Perhaps the total number of the squamous-cell type is even higher, since in some

Carcinoma of the urethra showing the presence of carcinoma just behind the glans. Two benign papillomas are seen just above the line of amputation.

of the case reports a diagnosis of carcinoma was made without stating the type of the cells. Normally, the prostatic urethra is lined with transitional epithelium and the penile urethra with columnar epithelium, only the fossa navicularis having squamous epithelium. The development of a squamous-cell carcinoma of the penile urethra can be

explained on the basis of metaplasia, as a result of which patches of leukoplakia form, which then undergo malignant degeneration. In the same manner, squamous-cell carcinomas occurring in other parts of the other urinary organs (in parts which do not normally contain squamous cells), such as the kidney pelvis and the urinary bladder, can be explained.

Columnar-cell carcinomas are quite rare. Only one of Preiswerk's collected cases belonged to this type, the one reported by Cabot. Of the cases in this series, none was columnar-cell carcinoma.

The papillary carcinoma is as rare as the columnar-cell variety. Preiswerk included only one case of this type, the one reported by Buday. Subsequently, cases of this type were reported by English.[3]

The case reported in this paper showed, besides a large papillary carcinoma, many benign papillomas. It may be stated with reason that one of the previously benign tumors had undergone malignant change, a somewhat analogous condition occurring in certain bladder tumors.

When adenocarcinoma is found, the consensus of opinion is that its source is most frequently in Cowper's glands; hence this type has been considered as primary in Cowper's glands with secondary invasion of the urethra. In rare instances, it may happen that the tumor originates in some of the urethral glands, in which case it would be classified as a true adenocarcinoma.

The carcinoma may spread along the mucous membrane of the urethra. As the tumor enlarges, the lumen of the urethra is encroached upon, and the symptoms of stricture supervene. At times it extends into the spongy bodies, and not infrequently grows outward so that a palpable mass is present. Infection is a concomitant of the tumor; hence suppuration ensues, and the clinical picture is that of a periurethral abscess. Later fistulas may form with escape of urine through the fistulous opening. In the course of the disease, there is an early involvement of the inguinal glands, record of which involvement has been stated in fifteen cases. In one case, involvement of the lumbar glands was found at necropsy (Montgomery[4]).

ETIOLOGY

Nothing is known regarding the etiology, although several factors have been mentioned as playing a predisposing rôle.

Age: The one constant factor of importance, as in other forms of carcinoma, is age, the tumor being essentially one of advanced or late adult life. The youngest patient whose case is reported was 38 years

3. English: Folia urolog. **1**:38-76, 1907-1908.
4. Montgomery: Med. Chron. **1**:190-193, 1901.

old (Röpke[5]), and the oldest, 74 (Peacock[6]). Between the ages of 30 and 39, one case has been reported; between 40 and 49, seven cases; 50 and 59, eighteen cases; 60 and 69, seven cases; 70 and 79, two cases, and the age was not stated in two cases.

Stricture: Several authors have mentioned a history of long-standing stricture. But, on the other hand, no history of stricture was found in a large number of cases, and many of the patients denied previous urethral infection, a matter reported by Barney,[7] Bierbaum,[8] Bonzani,[9] Hall,[10] Röpke, Montgomery, Platte,[11] Olivier and Clunet,[12] Romano,[13] Shattuck,[14] Scott[15] and Van Hook.[16] Stricture should be regarded as a factor only so far as it might produce metaplasia or leukoplakia of the urethral mucosa. An important point to remember, and one bearing on the subject of stricture, is that carcinoma may produce symptoms of stricture early in the course of the disease, and these symptoms may be wrongly interpreted.

Previous Venereal Disease: Since gonococcus infection of the male urethra is common, and carcinoma of this part of the body is rare, the assertion may be made that the gonococcus plays little, if any, etiologic rôle in the production of this disease. A positive history of previous gonorrhea is mentioned by Barney, Braasch and Scholl,[17] Conforti,[18] Deveze,[19] English, Menard,[20] Michon,[21] Ottow,[22] O'Neil and Rizzi.[23]

Regarding the part that preexisting local urethral conditions may play, it might be stated that in the case reported in this paper, the patient made the statement that twenty-five years previously many small papillary tumors were removed from the urethra by means of a curet; and many were found in the urethra after it was opened following the

5. Röpke: Arch. f. klin. Chir. **78**:233, 1905.
6. Peacock: Brit. M. J. **1**:138, 1910.
7. Barney, J. D.: Boston M. & S. J. **207**:790, 1907.
8. Bierbaum, K.: Inaug. Diss., Leipzig, 1912.
9. Bonzani: Folia urolog. **3**:491, 1908-1909.
10. Hall, J. B.: Ann. Surg. **39**:375, 384, 1904.
11. Platte: Med. Chron. **44**:94-98, 1906.
12. Olivier and Clunet: Bull. et mém. Soc. anat. de Paris **132**:735, 1907.
13. Romano: Riforma med. **28**:957, 1912.
14. Shattuck: Tr. Path. Soc. Lond. **58**:199, 1906-1907.
15. Scott: Bull. Cleveland General Hosp. **1**:30, 1899.
16. Van Hook: Chicago Med. Rec. **12**:303, 1897.
17. Braasch, W. F., and Scholl, A. J., Jr.: Ann. Surg. **76**:246 (Aug.) 1922.
18. Conforti: Clin. chir. **16**:315 (Feb. 28) 1908.
19. Deveze: Montpel. méd. **20**:193, 1905.
20. Menard: Bull. et mém. Soc. anat. de Paris **135**:394, 1910.
21. Michon: Ass. franç. d'urol. Proc.-verb. **15**:342, 1911-1912.
22. Ottow: Ztschr. f. Urol. **7**:30, 1913.
23. Rizzi: Ztschr. f. Urol. Chir. **7**:1 (July) 1921.

amputation. Whether these papillomas are to be considered as predisposing factors may be questioned. However, since papillomas in the bladder often undergo malignant change, there is no reason why the same phenomenon cannot take place in urethral papillomas.

Of further interest in this connection is the patient's statement that he attributes his present condition to the use of an injection into the urethra of Hartzell's solution. Since his condition developed so rapidly after the use of this injection, it seems reasonable to assume that the injection fanned the carcinoma into activity. Prior to the injection, the tumor may not have been noticed by the patient.

SYMPTOMS

First Stage.—Early in the course of the disease, the symptoms are practically nil. Often the first symptoms noted by the patient are those associated with stricture, which are caused by the obstruction produced within the lumen of the urethra by the tumor. Often the presence of a discharge first attracts the patient's attention to his local condition. Discharge was noted by Barney, Menard, Montgomery, Ottow, Peacock, Platte, Rizzi, O'Neil, Shattuck, Röpke, W. S. Scott, and in my patient. In O'Neil's case, there were gonococci in the discharge, and in Rizzi's case, the discharge was bloody. Definite obstruction to urination was noted by Bierbaum, English, Peacock, O'Neil, Rizzi and Tansini.[24]

Because of these symptoms in the early stage of the disease, the clinical picture is often confused with stricture; and the picture is still further complicated if the patient develops complete retention of urine after symptoms of urinary obstruction have been present for sometime. Retention of urine is not an uncommon symptom. Its presence was recorded by Barney, Bierbaum, Bonzoni, Deveze, O'Neil, Olivier and Clunet and Röpke. A history of stricture was mentioned by Anzilotti,[25] Barney, Bierbaum, Bonzani, Braasch and Scholl, Deveze, Montgomery, Platte, Paul,[26] Röpke, O'Neil and Scott.

However, one should not forget that the carcinoma produces an obstruction which is a malignant stricture. This fact should be borne in mind in eliciting the history, particularly when trying to establish a history of previous stricture.

Second Stage.—This stage of the disease may be characterized by the formation of a local tumor which may be the first symptom noted by the patient; hence this is the condition which causes him to consult a physician. Tumor is a common finding later in the course of the

24. Tansini, quoted by Bonzani (Footnote 9).
25. Anzilotti: Clin. chir., Milan **12**:60, 105, 1904.
26. Paul: Lancet **1**:1127 (June 20) 1885.

disease, as noted by Anzilotti, Barney, Bierbaum, Bonzani, Conforti, Deveze, English, Hall, Woods, Montgomery, Legueu,[27] Olivier and Clunet, Platte, Romano, O'Neil, Rizzi and Scott.

Third Stage.—As the disease progresses, infection supervenes, and there is superimposed the picture of a periurethral abscess, which has, indeed, nothing characteristic about it to distinguish it from any other type of periurethral abscess. If a patient with this type of abscess is brought into the hospital as an emergency case, the malignant nature of the condition is often not recognized, and this oversight can be readily understood. Periurethral abscess formation was present in cases reported by Barney, Hall, Lavenant,[28] O'Neil, Van Hook, Conforti, Deveze, English, Montgomery and Paul.

Fourth Stage.—Still later, the clinical picture is further complicated by the presence of fistulas. Urinary fistulas occurred in the cases reported by Anzilotti, Barney, Bonzani, Conforti, English, Hall, Montgomery, Legueu, Lavenant, Menocal,[29] Platte, Van Hook, O'Neil, Paul and Röpke.

As a rule, the opening of the fistula, or fistulas, is on the undersurface of the penis; but it may be found on any surface. Thus, in Legueu's case it was found on the dorsum of the penis.

TREATMENT

Just as soon as the diagnosis has been made, nothing short of a radical operation should be employed, namely, amputation of the penis with transplantation of the urethra and complete extirpation of the inguinal glands. This type of operation was carried out in the case reported in this paper. When the patient was last heard from, two years after the operation, there were no signs of recurrence.

If the diagnosis is made early, that is, when the tumor is small and before there is involvement of the regional lymph glands, one might resort to the use of radium.

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27. Legueu: Méd. mod. **15**:313, 1904.
28. Lavenant: Bull. et mém. Soc. anat. de Paris **129**:666, 1904.
29. Menocal: Rev. de med. y cirug. de la Habana **13**:133, 1908.

THE PATHOGENESIS OF PEPTIC ULCER*

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The etiology and pathology of gastric and duodenal ulcer have attracted the interest of investigators and clinical observers since the time of Cruveilhier, the more because of the fact that no satisfactory explanation has appeared. Many theories have been advanced, based on experimental investigation and clinical observation, and as many counter-theories and refutations, based on equally good material, have been called up in answer. The literature, therefore, abounds in probable theories of the causation, but everywhere one is impressed by the diversity of opinion and the variety of the avenues of approach employed by the investigators.

Our interest in the question was aroused by the observations reported from this laboratory by Ellis,[1] who found, in a few cases, hemorrhagic erosions and even perforating ulcers in the stomach and duodenum of dogs after the intravenous injection of toxins isolated from animals with a high intestinal obstruction. Erosions and ulcers were also seen after the removal of the suprarenal bodies, an occurrence which has been reported by Finzi,[2] Mann,[3] Friedman,[4] Silvestri,[5] and others. The interesting observation was also reported by Ellis,[1] that the content of the intestine after double suprarenalectomy, contains a poison identical with, or similar to, that found after high intestinal obstruction. The object of our experimentation was the attempt to produce ulcers by a functional high obstruction, by means of a blind duodenal pouch, and later by looping the upper intestine in various ways to produce chronic stasis.

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1. Ellis, J. W.: The Cause of Death in High Intestinal Obstruction, Ann. Surg. **75**:429 (April) 1922.

2. Finzi, O.: Ueber Veränderungen der Magenschleimhaut bei Tieren nach Nebennierenextirpation und über experimentell erzeugte Magengeschwüre, Virchows Arch. f. path. Anat. **214**:413, 1913.

3. Mann, Frank: Gastric Ulcer Following Removal of Adrenals, J. Exper. Med. **23**:203 (Feb.) 1916.

4. Friedman, G. A.: Probable Endocrine Origin of Peptic Ulcer, J. A. M. A. **71**:1543 (Nov. 9) 1918.

5. Silvestri, T.: Endocrine Origin of Gastric and Duodenal Ulcer, Policlinico **27**:673 (June 28) 1920.

It has been pointed out by many observers that the acute and chronic peptic ulcers are distinct from the superficial hemorrhagic erosions so often found in diseased and cachectic conditions. Our own observations were based on the assumption that a peptic ulcer is a circumscribed loss of tissue in the wall of the stomach or duodenum, surrounded by an area of reactive inflammation, and involving one or all of the coats of the intestine. Cruveilhier,[6] quoted by Bolton,[7] initiated the idea that chronic ulcer originated in acute lesions; and Rokitansky [8] subscribed to this view. Bolton believes that most acute ulcers heal rapidly, and that chronic ulcers result only from unhealed acute lesions. We accepted this view in our consideration of the problem, and advance the hypothesis that failure to heal, i.e., the chronicity of the ulcer, results from the continued pull of the muscle fibers about the margins of the ulcerated area. An ulcer which did not primarily extend into, or through, the muscularis would not become chronic, but would compare with the hemorrhagic erosion and readily heal.

In this connection, it is significant that Wilensky and Geist [9] reported that micro-organisms, isolated from human peptic ulcers and introduced into surgically produced ulcers in animals, have no influence in retarding healing or in the production of the chronic indurated ulcer. Ivy,[10] in describing the healing of the acute ulcer, points out the importance of the proliferation of connective tissue in the submucosa about the ulcer and the binding of the edge of the ulcer to the connective tissue base. Our conception of delay in healing is based on the assumption that the muscle fibers exert tension on the edges, preventing a reduction in size and the anchorage of the edge by connective tissue, and exerting the same influence on the healing process that constant movement exerts on a varicose ulcer of the leg.

Smithies [11] and Carlson [12] have produced evidence to show that high acidity of the gastric juice can have no influence in either the cause of ulcer or delay in healing. Bolton [7] and Friedman and Hamburger [13] secured delay in healing of acute ulcer by producing a partial

6. Cruveilhier: Essai sur l'anatomie pathologique en général **2**:54, 30.

7. Bolton, Charles: Ulcer of the Stomach, London, 1916.

8. Rokitansky: Lehrbuch der pathologischen Anatomie (Sydenham Society) **2**:31, 1849.

9. Wilensky, A. O., and Geist, S. H.: Experimental Studies in Production of Chronic Gastric Ulcer, J. A. M. A. **66**:1382 (April 29) 1916.

10. Ivy, A. C.: Studies on Gastric and Duodenal Ulcers, J. A. M. A. **75**: 1540 (Dec. 4) 1920.

11. Smithies, Frank: Significance of Etiologic Factors in the Treatment of Peptic Ulcer, J. A. M. A. **74**:1555 (June 5) 1920.

12. Carlson, A. J.: The Control of Hunger in Health and Disease, Chicago, University of Chicago Press, 1916.

13. Friedman, J. C., and Hamburger, W. W.: Experimental Chronic Gastric Ulcer, J. A. M. A. **63**:380 (Aug. 1) 1914.

pyloric stenosis. The former says that the retention of bacteria and acidulated food causes continued infection and irritation of the ulcer, with the migration of leukocytes to the site and necrosis of the base, thus hindering the growth downward of regenerating glands. The process then results in the excessive formation of scar tissue, over which the mucous membrane fails to grow. It seems reasonable that the stenosis sets up hyperperistalsis, with resultant pull of the muscle fibers about the margins of the lesion.

On the basis, then, that all peptic ulcers start as an acute lesion, some perforating, the majority healing, the others progressing to a

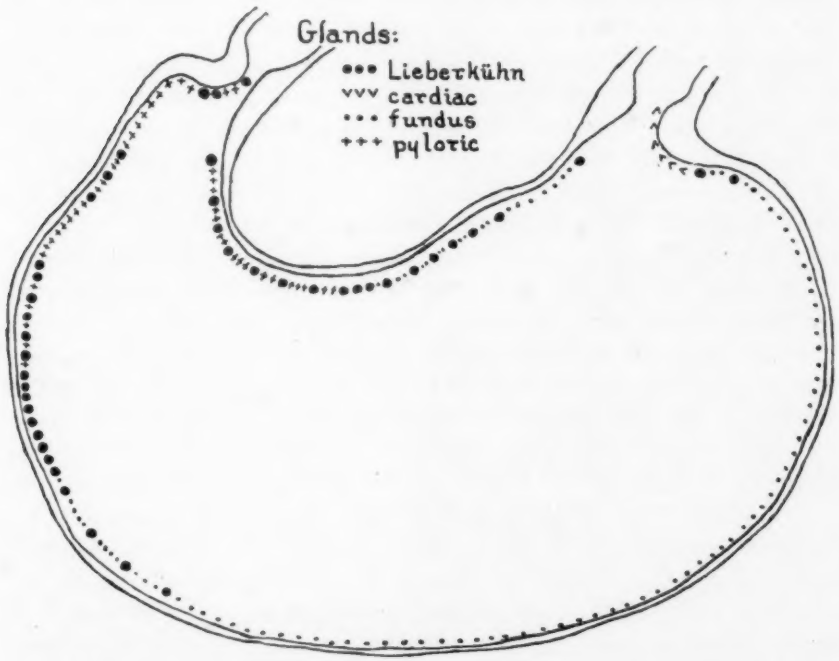

Fig. 1.—The arrangement of the glands along the two curvatures of the stomach (after Jouvenel).

chronic stage, we endeavored to reproduce acute ulceration by functional obstruction of the duodenum. The animals used were dogs, under strict conditions of etherization and asepsis. The operation consisted of section of the duodenum at varying distances from the pylorus, ranging from 10 to 30 cm., closure of the cut ends, and anastomosis between the stomach and the distal segment of the small bowel. The dogs that survived showed the characteristic hypertrophy and distention of the blind duodenal tube, with erosions and hemorrhages. In no case could a distinct peptic ulcer be found.

The next supposition was that a chronic functional obstruction could be used as a condition favoring a low-grade, chronic toxemia, which might induce the formation of ulcers. Moynihan [14] points out the existence of a condition which might be the expression of a toxemia of intestinal origin, preceding the clinical signs of ulcer: "About one fifth of the patients who have gastric ulcers complain not only of pain, but of great prostration, feebleness, or lassitude coming on just at the time when pain is due. On close inquiry, this most distressing symptom may be found to have preceded the experience of pain by weeks and months. The periodicity of the two is identical."

Lane [15] attributes the formation of ulcer to stasis in the duodenum. Rénon and Blamautier [16] report perforations of the small bowel in the course of rectal carcinoma, and attribute the cause of ulcerations preceding perforation to the prolonged stagnation of intestinal chyme and marked pathogenic activity of the bacterial flora of the intestine. Finally, Wilkie [17] states that chronic duodenal ileus is sometimes found in association with duodenal and gastric ulcer, and says that in cases of rapid collapse and death after the perforation of a duodenal ulcer, the signs of chronic duodenal obstruction should be looked for.

Our first technic, designed to produce an artificial duodenal stasis, involved the section of the reflection of peritoneum attaching the duodenum to the posterior abdominal wall; the twisting of the duodenum on itself to make a partial volvulus of mild degree, and the anchoring of the loop by suturing it to the stump of the peritoneal reflection. All the animals that recovered from the operation continued to live an apparently normal existence and showed no signs of ulceration in the intestine.

Following this, we attempted a partial functional obstruction by surrounding the duodenum, at a level of about 14 cm. from the pylorus, with bands of fascia dissected from the rectal sheath. The results in this series were as inconclusive as in the preceding cases. The walls of the duodenum proximal to the obstruction showed nothing more convincing than thickening and distention, with moderate infiltration of the mucosa by blood.

The next step was the production of a blind duodenal loop, with drainage through the distal segment, from a point above the loop or

14. Moynihan, B. G. A.: Essays on Surgical Subjects, Philadelphia, W. B. Saunders Company, 1921, p. 73.

15. Lane, W. A.: Operative Treatment of Chronic Intestinal Stasis, New York, Oxford University Press, 1918, p. 44.

16. Rénon, L., and Blamautier, P.: Ulcer and Perforation of Small Intestine in Case of Rectal Carcinoma, Bull. Acad. de méd., Paris **85**:715 (June 21) 1921.

17. Wilkie, D. P. D.: Chronic Duodenal Ileus, Brit. M. J. **2**:793 (Nov. 12) 1921.

from the stomach. The duodenum was sectioned at a level about 25 cm. from the pylorus and the proximal end carried back to be introduced into the duodenum above, by end-to-end anastomosis. The distal end was then introduced into the duodenum above the blind loop, in some cases; in other cases, into the stomach. The purpose of the operation was not to produce an absolute obstruction, but to establish a chronic functional obstruction, where stasis and a source of toxemia might be set up.

The results here were somewhat more encouraging. No dogs died of acute symptoms referable to gastro-intestinal disturbances. Dogs making complete and uneventful recovery were later killed and examined for evidences of ulceration. In only a few isolated cases were suggestions of an ulceration present. These were seen as localized hemorrhagic areas in the mucous membrane. In one case, this appeared in a suggestive location; i. e., on the lesser curvature in the pyloric antrum of the stomach. Our results must be acknowledged to be negative, so far as they concern the production of ulcer by a chronic, functional obstruction with stasis.

The question now arises whether the development of ulcer depends on a mechanism which disturbs the physiology of the intestine, be it obstruction, chronic stasis, embolism, vessel spasm, disturbance of innervation, or any one of the numerous theoretical explanations presented in the literature; or does the genesis of the ulcer depend on some anomaly seated in the anatomic structure of the intestine?

The characteristic peptic ulcer is a punched-out lesion that "looks as if something had dropped out," and is not primarily spreading, while the edges of the punched-out hole show no signs of involvement whatever. The fact that this hole is round inclines us to believe that the vessels are not involved, for even the end arteries of the mucous membrane do not supply a perfectly circular area. Likewise areas of ischemia would not result in a perfectly round lesion, nor disturbances of innervation. An area of infection might be perfectly round, but the adjoining edges must show reactive infiltration. It suggests that something had been in that site which was wholly distinct in itself, different from the surrounding parts, and of itself only susceptible to the influences that produced the lesion. The thought would be that some element exists in the mucosa which lends itself to the disturbances which promote necrosis and ulceration.

Recently, Müller [18] pointed out that peptic ulcer occurs in those regions of the gastro-intestinal tract of embryologic importance: in the stomach, at the region where there are islands of intestinal glands interspersed with gastric glands; in the esophagus where there are islands of gastric mucosa; in the duodenum and jejunum where there

18. Müller, P.: Beitr. z. klin. Chir. **123**:1, 1921.

are islands of pancreatic tissue; in Meckel's diverticulum where there are islands of gastric and pancreatic glands of incomplete anatomic structure, and, therefore, of abnormal function. Within such glands, he believes there can be an intracellular activation of digestive ferments, by virtue of which the glands themselves and their surroundings are digested.

Schäfer[19] in Quain's "Anatomy" records that true crypts of Lieberkühn occur in certain parts of the human stomach, similar in all respects to those of the small intestine. These are most numerous in a zone which lies between the area occupied by fundus glands and that occupied by pyloric glands, especially on the greater curvature.

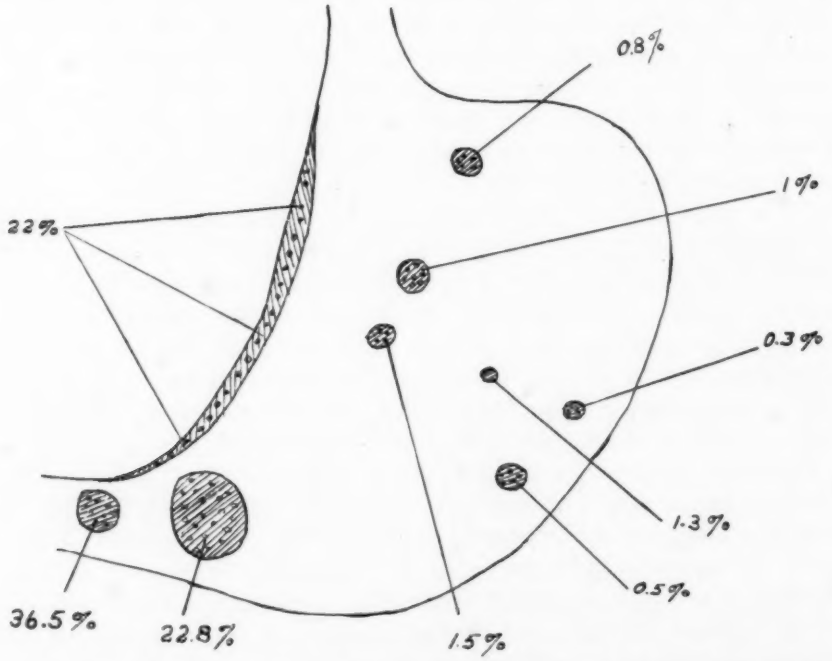

Fig. 2.—Areas of the stomach prone to ulcer, as determined by Krogius (from Dahl).

Attention was called to the presence of such crypts in the stomach mucosa by Schaffer.[20] Jouvenal[21] pointed out that the deepest cells of these crypts of Lieberkühn also exhibit granules similar to those described by Paneth in the intestinal glands, and says that such intestinal glands, found in the gastric mucosa near the pylorus, consist of normal cells and do not represent a metaplastic process.

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19. Schäfer, E. A., in Quain: "Elements of Anatomy," Ed. 11 **2**:533, 1912.
20. Schaffer, J.: Sitzungsb. d. k. Akad. d. Wissensch., Wien. **106**: 1897.
21. Jouvenal, F.: Répartition des Glandes de l'Estomac, J. de l'anat. et de la physiol. **42**:1, 1906.

Müller [18] studied fresh ulcers from the operating room; and in a case of perforating ulcer of a Meckel's diverticulum, he found that the diverticulum contained gastric mucosa resembling that of the normal pylorus and fundus. Extending his study to ulcers of the stomach, he found intestinal glands in the immediate neighborhood of the gastric ulcer in two of three cases. Müller discusses the proposal of Hammerschlag, that a transformation between gastric glands and intestinal glands occurs, which accounts for the presence of aberrant intestinal glands in the gastric mucosa. Müller asserts that the histology and embryology oppose this transformation, pointing out that the intestinal glands occur in sharply separated island formations, not mixed with gastric cells, and showing no transitional forms. Furthermore, such intestinal glands have been shown to contain beaker cells and distinct Paneth cells, as seen in the glands in their normal location.

Dahl [22] discusses an earlier report of Müller,[23] and calls attention to the distribution of areas of intestinal mucosa in the stomach, which he believes become subjected to the action of gastric juice, with results disastrous to the abnormally situated tissue. On this basis, he would explain differences in the susceptibility of persons and sexes, and the inclination of ulcer to follow in families. Dahl includes in his paper the two schematic drawings which are reproduced herewith, since they afford a picture of the areas of the stomach in which these aberrant islands occur, and compares these areas with the areas prone to ulcer, as determined by Krogius from a study of 391 cases. It is significant that histologists [24] do not report the occurrence of gastric mucosa in parts of the intestine below the duodenum and upper jejunum; correspondingly, the pathologists do not commonly find peptic ulcers below these levels.

The thought occurs that these glands being located in a foreign field are more subject to adverse influences by reason of the unusual environment. Intestinal glands normally accustomed to the alkaline reaction of the small intestine must be exposed to acid secretion when transplanted to the stomach. Their existence might not be jeopardized despite the unusual atmosphere unless some disturbing influence makes its presence felt through an alteration in the supply of blood to the part. The formation of ulcer after suprarenalectomy is well proved; after

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22. Dahl, P. R.: Sur la Pathologie de l'Ulcere, Arch. d. mal. de l'app. digestif. **10**:483 (April) 1920.

23. Müller, P.: Beitr. z. klin. Chir. **115**:560, 1919.

24. Broman, I.: Verhandl. d. anat. Gesellsch. **27**:14, 1913. Also Kaufmann, E.: Anat. Anz. **28**:465, 1906. Bensley, R. B.: Am. J. Anat. **2.** Schaffer, J.: Virchows Arch. f. path. Anat. **177**:181, 1904. Kull, H.: Anat. Anz. **41**:609, 1912. Arch. f. mikr. Anat. **77**:541, 1911. Trallero, M.: "Ueber das Verhalten der Muscularis Mucosae der Magenschleimhautinseln im Oesophagus," 1913, Dissert. med., Berlin.

suprarenalectomy, we also observe a steady fall in blood pressure. This is easily reflected in the stomach as an ischemia, which may lower the vitality and resistance of such intestinal gland cell rests to a degree which would not affect the normal mucosa, but would leave the aberrant tissue susceptible of destruction.

An ischemia or a stasis of blood to the part might well result in an asphyxiation, which in turn permits an autolysis, with the formation of an unknown substance, having the power of activating the proenzyme of the duodenojejunal glands intracellularly.[25] There would then follow an autodigestion, without any surrounding reactive inflammation. Thus, it is conceivable that the characteristic punched-out ulcer, looking "as if something had dropped out," would result, corresponding exactly to the isolated patch of aberrant intestinal glands, and no more; and "looking as if something had dropped out," because something had dropped out.

The question of the mode of formation in the duodenum now suggests itself. Deaver and Reimann,[26] in a study of recent ulcers, report two cases of duodenal ulcer removed at operation in which pancreatic tissue was found in the intestinal wall at the base of the ulcer, without any connection with the pancreas itself. Here the suggestion offers itself that a relic of embryologic defect is represented by the island of pancreatic tissue in that neighborhood of the intestine whence the pancreas developed, and that no disturbance arose from it until induced by such a disturbance as ischemia and asphyxiation which are supposed to occur in the case of gastric ulcer. Here there might be an activation of trypsin, with resultant autodigestion and a clean, punched-out ulcer corresponding only to the island of aberrant tissue.

Kaufmann [27] reports an accessory pancreas found at the tip of Meckel's diverticulum. Gegenbauer (1863), quoted by Broman,[24] called attention to an accessory pancreas in man, occurring usually in the jejunum and existing without any connection with the main pancreas. Broman saw a phylogenetic basis for an accessory pancreas in man, since in the lower forms the pancreas is represented by accessory glands distributed along the upper portion of the intestine.

The question in part becomes one of embryologic development and inherited tendency. Naturally the occurrence of peptic ulcers in the lower animals is rare. Turck [28] reported necropsies on 189 healthy and

25. Sweet, J. E.: The Surgery of the Pancreas, Alvarenga Prize Essay, 1915, Internat. Clinics **4**:293, 1915.

26. Deaver, J. B., and Reimann, S. P.: Surgical Treatment and Pathology of Gastric and Duodenal Ulcer, Surg., Gynec. & Obst. **32**:103 (Feb.) 1921.

27. Kaufmann, E.: Lehrbuch der speciellen pathologischen Anatomie, Berlin, G. Reimer **1**:641, 1911.

28. Turck, F. B.: Intestinal Venous Stasis, J. A. M. A. **67**:1784 (Dec. 9) 1916.

eighty-two diseased dogs, in which the findings of "peptic ulcer" were absolutely negative. Mann [3] listed a series of more than 200 normal dogs and cats in which "no lesion of the gastric mucosa was found at autopsy." Ivy,[29] in studying 900 dogs in his laboratory which had been subjected to two or three hours of ether for experimental purposes, found, in diseased and cachectic animals, only two marked ulcerations. On the other hand, Cohnheim [30] reported 1,086 cases of gastric or duodenal ulcer, being 7 per cent. of 15,500 patients in the course of twenty years.

It is conceivable that by generations of natural selection in the lower animals strains showing a tendency to peptic ulcer have been bred out, as, indeed, the commercial value of animal eugenics has led to the breeding out of all forms of inheritable defects; whereas in man, with no control of breeding and under medical and surgical care, "ulcer families" have been preserved to propagate their tendencies to abnormal cell-rests. Hence, we find the influence of age, sex and family exerting its impression in the etiology of the condition.

It is difficult to conceive that a bacterial embolism will affect only a small circumscribed round area and produce a characteristic ulcer in the face of the richly anastomosing gastric vessels. Mechanical abrasion and digestion by gastric juice are difficult of conception when we consider how neatly the injured mucosa recuperates following an operative procedure involving section of the entire gastric wall, even in the face of the high acidity which normally follows such trauma.[11] Section of the splanchnics, suprarenalectomy or the injection of highly toxic substances admittedly produce ulceration; but we would like to subscribe to the hypothesis of Müller, that these are contributing causes only; that the basic cause is the autolysis of islands of pancreatic and intestinal gland rests, attendant on the lowered vitality enhanced by the disturbance of the contributing causes.

This hypothesis would explain the findings reported by Ellis [1] of perforating ulcer in some instances following the intravenous injection of the toxin of high intestinal obstruction. Ellis concluded that the only explanation of the origin of the toxin consistent with his experimental results is that under the conditions of the high obstruction, of suprarenalectomy, etc., some factor activates the proteolytic proferment formed by the intestinal mucosa within the cells, instead of, as normally, outside the cell, in the lumen of the intestine. The necropsy on an animal dying from the effects of the intravenous injection of this toxin offers a very striking picture. The mucosa of the intestine, beginning sharply at the line where gastric mucosa and duodenal mucosa meet,

29. Ivy, A. C.: Contributions to the Physiology of the Stomach: LII. Studies on Gastric Ulcer, Arch. Int. Med. **25**:6 (Jan.) 1920.

30. Cohnheim, P.: Arch. f. Verdauungskr. **27**:241, 1921.

presents a condition of dark, velvet red, hemorrhagic infiltration. Islands of intestinal mucosa in the stomach would presumably show a similar effect, and, in addition, would be subjected to the digestive action of still another enzyme besides the one activated within the cells—the pepsin of the stomach.

In the consideration of human ulcer, it might not be amiss to point out, as regards due influence of age, sex, familial tendency and seasonal recurrence, the experimental fact that anaphylaxis in the dog presents an intestinal picture practically identical with that produced by the toxin of high obstruction.

The proof of this thesis might not lend itself to ready experimentation, but Müller's suggestion should be worth considering as an argument in favor of a more careful study, such as is seen in the report of Deaver and Reimann, of the bases and surrounding parts of ulcers, with an extension of the study to a careful microscopic examination of the nearby parts of the intestinal wall.

CALCIFICATION AND OSSIFICATION OF THE MENINGES

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Calcification and ossification of the meninges have held the interest of pathologists and anatomists for many generations, and the numerous workers who have investigated the subject have brought to light so much information concerning them that our present knowledge of this subject seems almost complete. However, the importance of the roentgen ray in the diagnosis of cranial pathology and the fact that calcification and ossification of the meninges are to be considered in the interpretation of all roentgenograms of the head make a reconsideration of this subject appropriate. Bony new growths in the dura are found frequently in the animal kingdom. They are present in the falx of the dolphin, the seal, the platibus *(Ornithorhynchus paradoxus)* and in the tentorium of cats, bears, horses and pachyderms.[1]

The first reported instance of ossification of the meninges in man was probably that of Antonius Cattus[2] in 1557. In 1761 appeared Morgagni's "De Sedibus et Causis Morborum," in which were described two cases of ossification of the dura. Morgagni thought the symptoms were due to ossification. In one case, that of an apoplectic, between the leaves of the falx cerebri was a piece of bone of moderate thickness which was three finger-breadths long by one and one-half finger-breadths wide. The other case was that of a syphilitic patient who had had severe headaches and convulsions during the last months of his illness. In the falx cerebri were five pieces of bone attached to one another, so that scarcely one third of the falx was free from bone. In 1859, Molas[3] reported finding a piece of bone "of large dimensions" between the layers of the falx. In 1866, Jeannerat[4] found that in not

1. Aschoff: Pathologische Anatomie.

2. Cattus, cited by Cruveilhier: Traité d'anatomie pathologique générale, Paris, 1849.

3. Molas: Pièce osseuse développée entre les deux feuillets de la faux du cerveau, Compt. rend. Acad. d. sc. **49**:299, 1859.

4. Jeannerat, Stanislas: Des ossifications de la dure-mère, Strasbourg, 1866.

less than sixty-four of 625 necropsies were the dura mater and falciform process the seats of ossification.

In these cases, eighty-eight pieces of bone were found in the falciform process, and thirty-nine were found in the remaining parts of the dura. The bone formations were of three types: first, the plate forms which numbered sixty-five, of which fifty-one were in the falx (the largest, 8 by 1.8 by 0.7 cm.) and fourteen in the remaining portions of the dura; second, the stalactite forms which numbered thirty-five, and third, the kernel or granular forms, which numbered twenty-seven.

The finding of many pieces of bone of considerable size has been reported. In Winkler's [5] case the bone measured 9.2 by 5.5 cm. In *Schmidts Jahrbücher der in- und ausländischen gesammten Medizin,* 1858, the presence of a plaque, 7 by 7 by 1 cm., is reported. Burle and Morel [6] report a case with a plaque shaped like an oyster shell in the anterior part of the falx, measuring 6 by 3 cm. In Jackson's [7] case, the anterior two thirds of the falx was ossified. The plate of bone resembled a scythe blade, was about 4 inches (10.6 cm.) in length, 1 inch (2.5 cm.) wide and one-half inch (1.27 cm.) thick at the anterior extremity, and one-half inch (1.27 cm.) wide and one-fourth inch (6.3 mm.) thick at the posterior extremity. The surfaces of the plate were rough. Cruveilhier has described complete calcareous metamorphosis of the posterior third of the falciform process.

The ossifications are generally found in the falx, alongside the superior longitudinal sinus, especially where the falx is inserted into the crista galli, less frequently in other parts of the dura and most rarely in the tentorium cerebelli. They are never found in the dura which covers the base of the skull.

These ossifications are not to be confused with the calcareous deposits in the form of psammoma bodies or corpora amylacea which, in varying stages of development and of varying sizes, are of common occurrence in the arachnoidea of man and of the lower animals. Cushing and Weed [8] believe that, associated with this process of calcification, a hyperplasia of the arachnoidal mesothelium is liable to occur. The deposits of lime salt are apparently laid down in cell bodies, and, in this membrane, can bear no relation to the blood vessels. The occurrence of true bone formation in the arachnoid of man should be regarded as a similar phenomenon, except that one may consider the process of ossification to be proliferative rather than degenerative.

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5. Winkler: Inaug. Diss.

6. Burle and Morel: Ostéome de la dure-mère, Lyon méd. **114**:1095, 1910.

7. Jackson ,W. R.: Med. News **54**:477, 1889.

8. Cushing, Harvey, and Weed, L. H.: Calcareous and Osseous Deposits in the Arachnoidae, Bull. Johns Hopkins Hosp. **26**:367, 1915.

Zanda [9] reports sixty-three cases of osteoma of the arachnoid, and he believes that the ossification arises from the direct transformation of connective tissue into bone tissue by the deposition of calcium salts. If this is so, calcification and ossification may be considered to be closely allied processes.

Ewing [10] notes that multiple nodules of bone are found in the cerebral and spinal arachnoid in a considerable portion of cachetic subjects, and especially in the insane and epileptic. He says that Zanda has found that these nodules form about small vessels through degeneration, exfoliation and sclerosis of the endothelial cells and connective tissue, which form hyaline, then osteoid, and finally bone tissue. From these nodules, the bone plates develop.

Borst [11] speaks of leptomeningitis ossificans as a process in the pia mater, characterized by irregular plates of osteoid substance. The bone plates are partly the product of chronic inflammation with secondary calcification and, possibly, are formed in part from the osteogenic tissue of the skull by separation.

In embryonal life, the dura functions as the periosteum of the inner surface of the skull and is capable as such of osteogenesis. It is not to be wondered, as Ribbert [12] remarks, that we frequently find bony deposits which generally do not appear as tumors but as irregular plates. He believes that such plates as are found in the falx cerebri are probably formed from islands of bone forming tissue which are left behind in the differentiation of the dura. These bone formations are not strictly to be designated as tumors.

Although there is no age at which persons are exempt from calcification and ossification of the dura, those of advanced years show a certain predilection. Koester [13] quotes Netter as describing a case in a 12 year old girl.

The exostoses on the inner side of the skull resemble closely the ossifications which appear in the dura mater. The latter result from increased bone production of the outer layers of the dura mater and early are fused with the skull. The meningeal exostoses, according to Virchow, are always separated from the bone by a fibrous layer, thereby preserving their heteroplastic character.

Calcification, by which is meant the mere deposition of calcium salts in distinction to the more complex process of ossification or actual bone formation, occurs in the human body under varying conditions and in

9. Zanda: Zieglers Beitr. z. path. Anat. **5**, 1889.

10. Ewing, James: Neoplastic Diseases, Ed. 2, Philadelphia, W. B. Saunders Company, 1922.

11. Borst: Die Lehre von den Geschwülsten, p. 166.

12. Ribbert: Geschwülste Lehre, p. 200.

13. Koester: Knochenbildungen in den meningen, Würzburg, 1903.

various places. Nunokawa's [14] classification is a very serviceable one. He divides the calcifications as follows:

1. Calcification due to insufficient nutrition:
   - (*a*) of thrombi,
   - (*b*) of hematomas,
   - (*c*) of tumors of the skin,
   - (*d*) of the insertion ring of the bicuspid value in senility,
   - (*e*) of the artery walls in arteriosclerosis,
   - (*f*) of hyaline degeneration of blood vessels,
   - (*g*) of unattached appendices epiploicae,
   - (*h*) of the kidneys and the seminal vesicles in senility,
   - (*i*) of placental villi,
   - (*j*) of inflamed indurations of the pleura and peritoneum,
   - (*k*) of chronically inflamed heart valves,
   - (*l*) of different tumors.
2. Calcification after sudden necrosis:
   - (*a*) muscle fibers near a wound,
   - (*b*) muscle fibers made necrotic by ligature,
   - (*c*) anemic heart infarcts,
   - (*d*) ganglion cells of the brain and cord killed by trauma,
   - (*e*) kidney, after certain poisons (mercuric chlorid),
   - (*f*) lithopedion.
3. Calcification metastases (Virchow).

In this class, local disturbances of bone cause the freeing of calcium salts which are deposited elsewhere, notably in (*a*) the endocardium; (*b*) the pachymeninx cerebralis; (*c*) the stomach or intestine.

4. Calcium deposits in thickened exudates, foreign bodies in the cavities lined by mucous membrane (as in the nose, tonsillar crypts, bronchi, intestine and the vagina).

5. Unexplained calcifications, such as those in the lung and muscle.

Virchow classifies bone tumors into: (*a*) the hyperplastic, which are entirely bone tumors arising from the osseous system and in which an abnormally increased production of bone is added to bone which is already present, and (*b*) the heteroplastic bone tumors which are entirely ossifications, arising in organs of the body not in conjunction with normal bone, and which in no way naturally tend to bone formation.

Pathologic calcification is of two types chemically: (1) that which appears in tissue poor in fats and which corresponds in its synthesis to that of "bone soil," that is, it contains tricalcium phosphates and calcium carbonates in a quantity relationship similar to that in the bone, and (2) calcification of fat tissues (as lipomas), which, on the other hand, can follow the deposition of calcium soaps.

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14. Nunokawa, K.: Ueber Verkalkung der Pachymeninx bei Usura Cranii, Virchows Arch. f. path. Anat. **198**:271, 1909.

In order to ascertain how well the blood and lymph are able to function in the transportation of calcium phosphate, Hofmeister [15] added, to the blood serum, calcium salts and sodium phosphate in varying quantities and in varying alkalinities, and found that the blood serum was able to hold in solution much more of the resultant calcium phosphate than it was possible to dissolve in water. This power of holding in solution diminished with dilution. In albumin concentrations, such as are found in the tissue lymph, the power of holding this salt in solution is very much diminished.

The researches of Dr. Tanaka have demonstrated the solubility of ivory plates, bone plates and tooth fragments in fluids (water, salt solutions and blood serum) which have been saturated with carbonic acid. His work confirms the previous researches of Maly and Donat, Flesch and Tillmans that the solvent power is very considerable.

In water and salt solution, a square centimeter of bone surface at 37 C. gave off about 0.15 gm. of substance (mostly calcium phosphate).

For explanation of the resorption and erosion of adult bone, it is sufficient to say that the tissue lymph of the osteoclasts and other cells which bathe the bone tissue contains so much free carbonic acid that it exercises a dissolving action.

If tissue fluids which hold calcium phosphate dissolved by carbonic acid lose their content of free carbonic acid through giving off to the outside or through combination with an alkaline medium, it is possible that their active fluid movements or higher albumin content does not prevent the deposition of calcium phosphate.

If a slow stream of dilute soda solution is allowed to run through a tube with semipermeable walls, as, for example, a reed, and if this tube is submerged in a solution of calcium phosphate which has been dissolved by carbonic acid, the wall of the tube becomes encrusted, in a characteristic manner, partly with amorphous and partly with spherical, formed calcium phosphates. This may account for the repeatedly observed calcification of the interior of arteries and the walls of capillaries.

In the same way may be explained what otherwise would be very difficult to understand, namely, the appearance of metastatic calcification in lung alveoli and stomach mucosa from the giving off of acid (carbonic acid, hydrochloric acid), which occurs in these places. In this connection, it is noteworthy that the metastatic calcium deposits in the mucosa of the stomach closely correspond to the extent of the investing cells.

Experiments of Tanaka have shown that an oversaturation of the tissues with calcium salts, even in the absence of predisposing pathologic changes, tends to cause calcification.

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15. Hofmeister: Experimentelles über Gewebes-Verkalkung, München. med. Wchnschr. 1909, p. 1977.

Injection of chlorids, lactates, acetates, carbonates, or phosphates in the peritoneal cavity or in the tissues of rabbits, dogs, etc., have, as a result, more or less extended and occasionally widely dispersed the calcification of the neighboring tissues, even, now and then, into widely separated regions. Moreover, there appears directly in the peritoneal cavity or in calcium carbonate which has been brought through a reed, a gradual transformation into phosphates.

A simple explanation of the deposition of calcium soaps in necrotic or autolytically changed fat tissues is now available. This may also readily be imitated in experiments. If one takes fat tissue, and, at 37 C., treats it with a stream of calcium phosphate held in solution by carbonic acid, in a short time there will be macroscopic and chemical evidence of calcified fat tissue. This is due to a gradual fat splitting, whereby the liberated fatty acids act as calcium binders.

One cannot yet definitely explain the frequently observed calcification of dead or undernourished tissues.

Wells [16] divides pathologic calcification into two forms: one a precipitation of calcium in secretions and excretions of the body; the other, the deposition of calcium salts in the tissues themselves. He believes that normal ossification and pathologic calcification (except metastatic calcification and the calcification of fat necrosis and other areas of necrotic fat tissue) depend more on physicochemical factors and variations in the carbon dioxid concentration than on the presence of chemical precipitates in the tissues. An increase in the alkalinity of the fluids makes the calcium salts decidedly less soluble, and when there is excretion of acids, the fluids in the substance of the organs are correspondingly alkaline.

The following considerations are concerned with the appearance of calcification in degenerating tissues: (1) the increased alkalinity or decreased carbon dioxid in the degenerating tissues, causing precipitation of the inorganic salts in the fluids seeping slowly through them; (2) utilization of the protein of the fluids by the starved tissues so completely, because of its flow through them, that the calcium cannot be held longer in solution; (3) the formation within the degenerating area of a substance or substances having a special affinity for calcium; (4) production of a physical condition favoring the local absorption of salts, the least soluble salts accumulating in excess.

Thiroloix and DuPasquier [17] report the case of a patient dying from a cerebral hemorrhage in whom there was an ossified plaque, 8 by 6

16. Wells, H. G.: Chemical Pathology, Ed. 3, Philadelphia, W. B. Saunders Company, 1918, p. 435.

17. Thiroloix and DuPasquier: Ossification de la dura-mère: mort par haémorrhagie cérébrale, Bull. Soc. anat. de Paris **68**:38, 1893.

inches, in the dura mater. They believed that the dura took on an osteogenetic function because of the congestion.

Microbic agents are thought by some to play a part in the causation of certain varieties of calcification of the meninges. Lutembacher [18] reports a case of a woman dying of typhoid fever, in whom the tiny fibrous plaques adherent to the visceral surface of the spinal arachnoid contained typhoid bacilli. In another instance,[19] fibrous plaques were found which enclosed the meningococcus.

While authors agree that the bone is in relation to the dura, there is a difference of opinion as to whether or not it is primary in the dura, as Virchow, Borst and Kaufman contend, or whether it develops first in the arachnoid, and then as fully developed bone is attached to the dura (Cruveilhier, Otto).

The causes have been given as: (1) inflammation caused by trauma (Elsner); (2) disturbance of nutrition (Virchow); (3) displacement of osteogenetic tissue (Ribbert and Borst); (4) irritation of the dura caused by displaced particles of the skull, or, more often by hyperemia, both of which result in ossification, and the etiology was reported as unknown by Förster, Schmaus and Borst.

Weber [20] summarizes his study of the subject in part as follows:

1. Ossifications of the dura belong not to the class of genuine tumors but to inflammations.

2. Such ossification, especially when it occurs in the sinus wall, as a rule, probably on account of disturbances of nutrition, appears first in the outer parts of the dura and then gradually spreads out centrally.

3. It may further thrombosis of the superior longitudinal sinus.

The significance of ossification and calcification of the meninges has aroused the speculative interest of observers for a long time. The earlier men, quite naturally, on finding an ossified falx at necropsy, sought symptoms, generally mental, to attribute to it. But before long, it became apparent that the calcification occurred in mentally sound persons, and, moreover, were not even common in the insane.

In Wood's [21] case, there were four pieces of bone in the falx, the largest three-fourths by one-fourth inch (19 by 6.3 mm.), and this author thought that the paralysis of the patient's left arm and leg was of interest in connection with the ossification of the falx. In a case in

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18. Lutembacher, M. R.: Ann. de méd. **5**:148, 1918.

19. Lutembacher, M. R.: Plaques fibreuses de l'arachnoide en clavant des meningocoques, Ann. de méd. **8**:51, 1920.

20. Weber, Richard: Ueber das "Osteoma durae Matris" und seine Beziehungen zum Sinus longitudinalis superior, Zürick, Gebr. Leemann & Co., 1912.

21. Wood, H. C.: Bony Deposit in the Falx, Softening in the Pons Varolii, Phila. M. Times **12**:350, 1881-1882.

which there was "irritation of the nerve centers," Morgan [22] thought such symptoms were accounted for by a small plate of bone in the right side of the falx cerebri. In a case of mania, Sneve [23] found a piece of bone, 4 by 1 cm. by 6.25 mm., between the layers of the falx cerebri.

More lately, the conception of calcium metastases, to which Virchow's name is attached, has attracted study and interest. Wells calls attention to the fact that in conditions with much destruction of bone, as osteomalacia, caries and osteosarcoma, deposits of lime salts have been found distributed diffusely in various organs, particularly in the lungs and stomach. As much as 13.38 per cent. of the dry weight of the lung and 12.15 per cent. of the kidney have been found as calcium monoxid in such a case.[24]

In usura cranii, the condition of circumscribed osteoporosis of the skull, deposits of calcium have been found in the dura. Chiari [25] and Orth [26] called attention to this in 1909, and the latter reported the case of a child, aged 11½ years, who had a round-cell sarcoma of the pedunculus cerebri, in whom the dura, posteriorly in the midline, contained hard white plaques. Nunokawa,[14] a pupil of Chiari's, made further investigation along this line. In another communication, Orth [27] mentions the frequency with which the calcium deposits of the dura are found in cases of bone resorption of the skull.

Allenbach [28] further elaborates the theory that the calcium salts set free by the osteoporosis are deposited metastatically elsewhere. He reports two cases in which at operation white calcium deposits in the pachymeninx were noticed by the operators, who, accordingly, felt justified in making a diagnosis of usura cranii. Allenbach maintains that calcification of the pachymeninx is always a sign that the brain pressure must have acted long enough and strongly enough to call forth a usure of the skull, thereby making possible a calcium metastasis to the dura. Schmidt [29] noted that there were macroscopic calcium deposits on the outer surface of the dura, and that, in the corresponding places, the skull was atrophied and rough, a condition which occurs where most pressure is exerted. This writer studied the normal dura

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22. Morgan, C. W.: Case of Osseous Formation of the Dura Mater, Australasian M. Gaz. **4**:35, 1884.

23. Sneve, H.: An Unusual Osseous Growth in the Falx Cerebri, Med. News **54**:320, 1889.

24. Virchows Arch. f. path. Anat. **197**:172, 1909.

25. Chiari: Zur Kenntnis der path. Verkalkung, München. med. Wchnschr. 1909, p. 481.

26. Orth: Charité-Ann. **33**:376, 1909.

27. Orth: Bemerkung zu Allenbruchs Arbeit, Virchows Arch. f. path. Anat. **205**:274, 1911.

28. Allenbach: Virchows Arch. f. path. Anat. **205**:474, 1911.

29. Schmidt, H.: Virchows Arch. f. path. Anat. **115**:142, 1914.

by a special microscopic technic and found that, after the age of 17 years, it contained calcium, and, after 40 years, there was as much calcium in some duras as there was in brain tumors.

Another writer [30] notes that calcification of the pachymeninx not only is found in usura cranii but also is sometimes quite widespread in inflammatory growths.

There has been to date no roentgenographic study of calcifications of the meninges. By means of stereoscopic roentgenograms of the skull it is possible to demonstrate calcified plaques in the falx and perhaps even in the parietal dura. A roentgenogram of the head of the cat reveals very plainly diffuse calcification of the falx with the sharp curving inferior border. In the case reported herewith, in which operation was performed by one of us (A.E.H.) for tumor of the brain, the roentgen ray revealed shadows which doubtless were due to calcification in the falx.

REPORT OF CASE

History.—M. B., a colored woman, aged 38, was admitted to the medical service of Dr. Robert B. Preble at St. Luke's Hospital, Chicago, Oct, 1, 1921. The complaints on admission were: (1) frontal and occipital headaches which had been very severe, having lasted from five to ten minutes at a time and having occurred frequently every day for the previous three weeks; (2) vomiting, which began two days after the headaches started and which was not projectile in character; (3) dizziness; (4) double vision, and (5) numbness, weakness and muscular twitchings of the right arm and leg. The patient had had three miscarriages. The left breast had been amputated two years previously for "cancer." Otherwise, the history was negative.

Examination.—This revealed: (1) slight rigidity of the neck; (2) double neuroretinitis, with hemorrhages from the retinal vessels (Dr. Frank Brawley); (3) marked choked disk, slight paresis of the right side of the face, right hemiparesis, inclination to fall toward the right, a right palpebral fissure narrower than the left (Dr. George W. Hall). The blood Wassermann reaction was negative. The spinal fluid examination showed: (*a*) negative Wassermann reaction; (*b*) positive Pandy test; (*c*) faintly positive Ross-Jones test. Examination of the urine was negative except for a trace of albumin. The roentgen-ray examination of the skull by Dr. E. L. Jenkinson was reported thus:

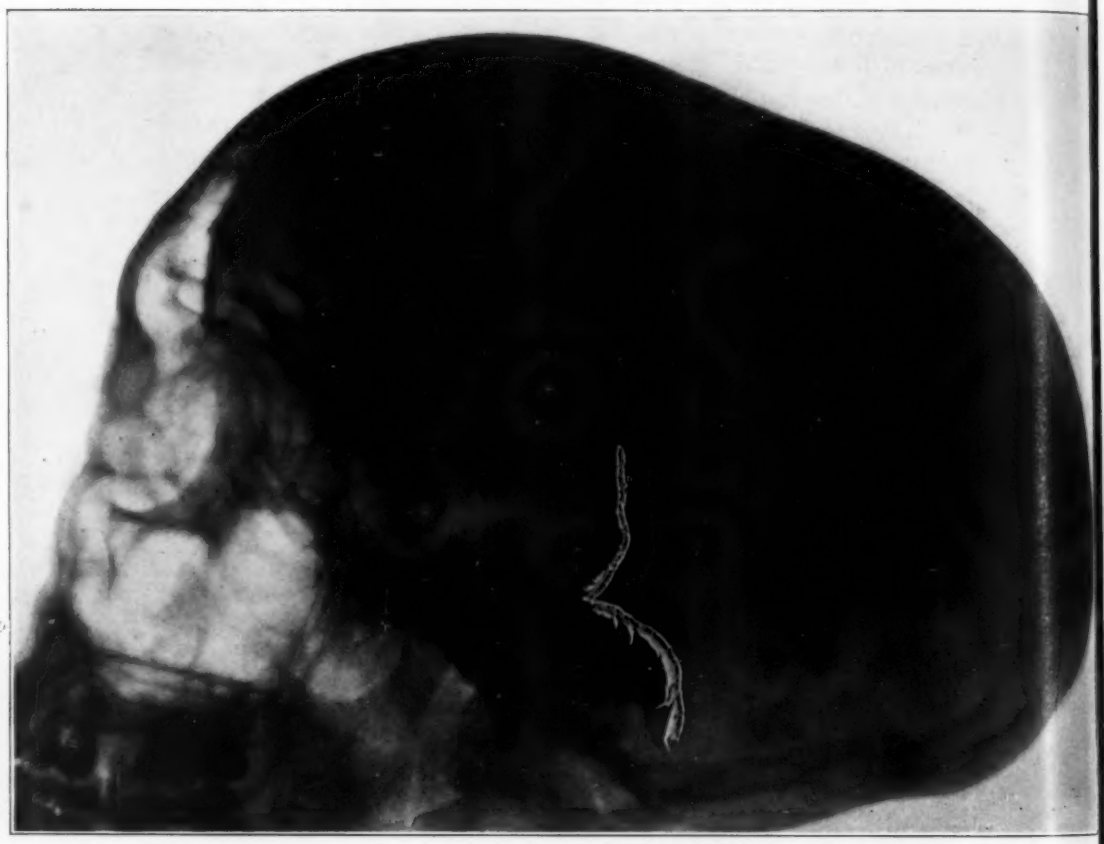
"Extending downward from the frontal bone are many irregular areas of increased density which may be due either to new bone involving the internal table of the frontal or to ossification in the falx [as in the accompanying illustration]. There are areas of ossification which have no connection with the frontal bone. I believe the areas of ossification are located in the median line, as there is no difference in the size of the areas taken in the right and left lateral positions. If the deposits were in the frontal bone there should be definite changes in their sizes when the right and left sides are exposed. In other words, if the deposits were located in the right frontal they would be smaller when the right side was near the film and vice versa. The plaques

30. Saltykow, S.: Zur Kenntnis der Duraverkalkung bei Schädelusur, Verhandl. d. deutsch. path. Gesellsch. **17**:521, 1914.

ranged in size from 0.5 to 1 cm. in diameter. The total area involved measured about 6 by 3 cm. The ossification seemed confined to what we believe is the anterior third of the falx. The tentorium cerebelli shows no areas of ossification.

"Summary: Ossification of the falx cerebri."

Operation.—The patient's condition became worse, and Dr. Hall advised a left subtemporal decompression. Oct. 13, 1921, the patient was transferred to the surgical service of Dr. Albert E. Halstead, and a left subtemporal decom-



Lateral view of skull, showing the irregular, curved shadows just posterior to, and above, the frontal sinus, which were interpreted as being caused by calcified plaques in the falx cerebri.

pression, accomplished by elevating a bone flap, was performed by Dr. Halstead. The dura was opened, and no hemorrhage was found. Following this operation the patient improved steadily, was up and about, Oct. 16, 1921, and was discharged, Oct. 24, 1921.

Readmission.—She was readmitted to St. Luke's Hospital, Jan. 21, 1922. Her complaint at this time was inability to talk properly and paralysis of the right arm and leg. Examination disclosed a pulsating cerebral hernia in the left temporal region. There was complete right hemiplegia, including the facial

nerve. She was also suffering from aphasia. The left palpebral fissure was wide, and the eyeball seemed to protrude. There was a neuroretinitis, with papillitis of 2 diopters. An attempt to inject the lateral ventricles with air through a trephin opening was made; but the patient's condition became so serious that the operation was discontinued. The patient was unimproved and was discharged, March 11, 1922.

Subsequent Note.—The social service department found that the patient died at home, July 11, 1922. Necropsy was not performed.

CONCLUSIONS

From the foregoing considerations the following conclusions may be deduced:

1. Ossification and calcification of the meninges are not rare.

2. Areas of ossification may arise from : (*a*) the retention of the osteogenetic function by islands of dura; (*b*) an inflammatory process.

3. Areas of calcification may arise as: (*a*) the result of a not far distant absorption of calcium of the skull and its subsequent deposition in the meninges; (*b*) the result of an inflammatory process.

4. In cases of suspected brain tumor, the demonstration by means of the roentgenogram of calcification of the meninges is an additional evidence of the existence of a brain tumor exerting a circumscribed pressure on the skull.

5. The chance finding of ossification or calcification in roentgenograms of the head affords the occasion for an intensive neurologic study of the case in question.

TWENTIETH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY *

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CONGENITAL CONDITIONS

Congenital Dislocation of the Shoulder.—Todd,[1] in examining the shoulder joint of 730 subjects in the dissecting room of Western Reserve Medical School, found three cases of what he terms posterior congenital dislocation of the shoulder of the subacromial type. The chief deformity is in the scapula, the glenoid being rotated backward on the blade. There is a drooping of the acromion, and while the humeral head remains in articulation with the glenoid, its configuration may be changed. In this series, there were no congenital dislocations of the radius or of the femur.

Congenital Defects of the Spine and Ribs.—Sever,[2] in a study of more than 2,000 roentgenograms of the spine, taken at the Boston Children's Hospital for various reasons, found twenty-two cases of cervical ribs, in nine of which there were other deformities of the vertebrae or ribs. In twenty-six cases, there were defects of the vertebrae in the dorsal region, with rib defects and scoliotic deformities. Lack of fusion of the spinous processes and laminae cannot be regarded as abnormal until after the seventh year, when they normally fuse. Eighteen true defects of the lumbar spine and sixteen of the sacrum were discovered. These two latter defects were often combined.

Reduction of Congenital Dislocation of the Hip.—Goyder [3] has employed with satisfaction a push rather than a pull method of reduction of congenital dislocation of the hip. He believes less trauma and more perfect control are possible by this procedure. In the manipulation of a left hip, the sacrum is placed on a sand bag; and the pelvis

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* This Report of Progress is based on a review of 516 articles selected from 610 titles dealing with orthopedic surgery, appearing in medical literature between May 21, 1922, and October 20, 1922. Only those papers which seem to represent progress have been selected for note and comment.

1. Todd: Ann. of Surg., July, 1922.
2. Sever: Boston M. & S. J., June 15, 1922, p. 799.
3. Goyder: Brit. M. J., Sept. 16, 1922, p. 497.

is steadied by an assistant. The left knee and hip are flexed, the hip as fully as possible, until the knee lies as nearly as possible by the side of the trunk. The right hand of the operator is placed on the patient's left knee, and the operator strives (1) by downward pressure, to push the head toward the acetabulum, and (2) by rotation of the knee, to direct the movements of the head. The palm of the operator's left hand is placed against the great trochanter, with the thumb in front and the fingers on the neck of the femur behind. While the downward pressure on the knee is maintained with the right hand, an attempt is made, by lifting with the fingers of the left hand and depressing the right hand, with or without rotation, as is demanded, to place the head definitely below the acetabulum. It may require a little time to stretch the tissues below the acetabulum, but no great force is necessary. The last procedure is to relax the pressure on the knee while the forward position of the head is maintained; and the head will be found to sink gently into the acetabulum. Often, it will be found at this stage that reduction has become possible by the more usual method of lifting the head of the femur over the posterior lip of the acetabulum.

Davis method: Merrill,[4] having worked for many years with the late Dr. G. G. Davis of Philadelphia, feels that his method of reduction of congenital dislocation of the hip has the great merit of gentleness and perfect control, and that familiarity with the method should be more general. The essentials and order of the procedure are: direct pressure, flexion, internal rotation, abduction, external rotation. The patient is placed prone on a flat, firm table, evenly padded, and the leg is allowed to hang downward close to the side of the table. There are two phases of the procedure: 1. The pelvis is fixed by one assistant, while another grasps the foot and knee, flexing the thigh toward the axilla, internally rotating the thigh and making pressure toward the head of the femur in line of its axis, keeping the thigh close to the trunk. The operator directs these movements and makes pressure downward on the trochanter toward the tuberosity of the ischium. 2. When the head has been pushed to the posterior brim of the acetabulum, the operator grasps the knee and trochanter, extending and abducting the thigh toward the transverse plane of the pelvis, and, within the bounds of safety, making pressure on the trochanter toward the acetabulum, at the same time gently rotating the thigh inward and outward. If these procedures are not successful, the first phase is repeated until the head is pushed downward to the obturator groove. It is then carried upward through the cotyloid notch by means of external rotation and extension. The plaster cast is applied in what seems to be the most stable position for the individual case.

4. Merrill: J. Bone & Joint Surg., Oct., 1922, p. 789.

Denucé's Method: Adams,[5] after a personal observation of Denucé's methods and results at Bordeaux, has been greatly impressed by both. The procedure closely resembles the manipulation of Ridlon, except that tight adductors, which have been powdered with talcum, are gradually stretched by gentle stroking by the operator's palm and thenar eminence downward in the longitudinal axis of the thigh. No violence or great force is used in any stage of the manipulation. The patient lies on his back. As the adductors become relaxed, the thigh is brought up to right angle flexion and full abduction. No attempt is made to reduce the hip until the thigh will lie on the table at right angles to the trunk. The operator stands beside the affected hip and flexes the thigh, the knee being carried across the trunk in the direction of the opposite axilla, and the thigh being firmly pressed down upon the anterior body wall. The knee is held high in this position, a slight push being exerted in line with the axis of the femur. The fingers of the operator's other hand are placed under and around the head, neck and trochanter, lifting upward and pressing the muscles in between the head and the side of the pelvis.

In the next step, circumduction, the knee is carried across the trunk toward the affected side and then slowly downward to the surface of the table, and gradually abducted. During this procedure, the fingers behind the head are held firmly in place and exert a lifting force. Circumduction is slowly continued until the thigh is brought to a right angle with the trunk and lies flat on the table, the assistant meantime holding the pelvis firmly by pressure on the opposite iliac crest and anterior superior spine. The head is now felt to come gradually forward and lodge in the acetabulum. The term "oozes in" has been applied. The limb is ordinarily put in 90 degrees of flexion and abduction, the plaster extending from the nipple line to the ankle. The child is sent home and kept recumbent for three months, when, frequently, the cast below the knee is removed. After six months, the cast is cut into a lower and an upper half, and daily applications of hot sand bags are made to the anterior surface, while the child lies in the posterior shell. Heliotherapy is instituted. The child is encouraged to lift and straighten the leg voluntarily. At the end of the ninth month, the whole cast is removed to allow active motion and exercises of the glutei. As an aid in these exercises, the parents construct a water tight box and fill it with a brine solution so buoyant that the child bobs up and down in it. Weight bearing is allowed one year from the time of reduction.

Results of Reduction of Congenital Dislocation of the Hip.—Fairbank[6] estimates that an anatomic cure ought to be obtained by manipu-

5. Adams, Z. B.: J. Bone & Joint Surg., July, 1922, p. 523.
6. Fairbank: Brit. J. Surg., July, 1922.

lative reduction in about 75 per cent. of the unilateral and 50 per cent. of the bilateral cases in the early years (before the age of 6). In the vast majority, function, for many years at least, should be excellent. Some successes may be obtained by a second manipulation if the first fails, but he recommends an open operation within two or three weeks of the second attempt. The operation which he advocates is not an open reduction, but an attempt to make an upper lip for the shallow acetabulum without opening the joint. Open reduction should be reserved for those cases in the middle age group in which manipulative procedures have failed. In only carefully selected cases should open operation be performed after the age of 6 years.

Evans [7] has been impressed with the bad results which follow attempts by untrained surgeons to reduce congenital dislocations of the hip.

Report of Commission to Investigate the Best Methods of Treatment of Congenital Dislocation of the Hip.—The commission appointed by the American Orthopaedic Association, consisting of Goldthwait, Adams and Willard,[8] emphasizes the importance of careful study of the individual case before and after operation. The depth and conformation of the acetabulum, the shape of the femoral head and the angulation of the neck, as well as the axis of the epiphyseal cartilage, are of great importance in prognosis. The commission is convinced that the violence used in reduction bears a direct relation to the large number of abnormally shaped heads which must make good permanent function less likely. They believe that the methods of Ridlon and Denucé are the best thus far devised. They consider that, of the machine methods, Hibb's is the least objectionable. It resembles the procedures of Ridlon and Denucé except that the forward movement of the head, once it has been drawn down sufficiently, is accomplished mechanically instead of manually.

Bernstein [9] reports the successful open reduction of a congenitally dislocated hip in a woman of 21, with 4 inches (10 cm.) of shortening. He removed a portion of the outer lip of the iliac crest and fastened it by bone screws over the upper lip of the acetabulum, in order to deepen the cavity. The gluteal muscles and the capsule were freely stripped down and out, and great force was necessary in traction to accomplish the reduction. The after pain was so severe that Bernstein recommends preliminary injection of the sacral canal to overcome the distressing symptoms of nerve stretching.

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7. Evans: Brit. J. Surg., July, 1922.

8. Goldthwait, J. E.; Adams, Z. B., and Willard, DeF. P.: J. Bone & Joint Surg., Oct., 1922, p. 821.

9. Bernstein: J. Bone & Joint Surg., July, 1922, p. 481.

[Ed. Note.—It seems to us interesting that strong advocates of the push rather than the pull method of reduction of congenital dislocation of the hip are coming forward. The procedures of Groyda, Schanz, Davis, Ridlon and Denucé have many points of similarity. The most striking common point is their gentleness and avoidance of trauma, not only to the femoral head, but also to the soft parts. The advantage of great gentleness is also strongly brought out by the report of the Commission of the American Orthopedic Association. Nothing can be more impressive than a series of roentgenograms of the femoral heads and acetabula taken years after successful manipulative reductions. A large proportion show changes in shape which must at least threaten permanent perfect function. Trauma at the time of reduction seems the most likely cause for these changes.]

The Production of Temporary Paralysis in Difficult Cases of Congenital Dislocation of the Hip.—Robertson [10] advocates a method of producing temporary relaxation of the adductor muscles, the contraction of which he considers as much more important than the hour glass contraction of the capsule or the tension of the psoas muscle as an obstacle to reduction. Two weeks before the attempted reduction, he exposes the obturator nerve and ligates it with silk suture tied in a bow knot, the ends of which protrude through the wound. Beneath the first tie of the bow, another strand of silk is placed, the ends of which also protrude through the wound. The ligature may thus be untied and removed after the wound is closed. Constriction of the nerve maintained in this way for from one and one-half to two hours may be expected to produce a paralysis lasting for six months. The tendon of the adductor magnus is also divided at the adductor tubercle, since it has a separate nerve supply. He has employed the method also in securing alinement in certain difficult fractures of the long bones.

[Ed. Note.—The Editors have had no experience with this method, but do not recommend it *a priori*. They are not convinced that the contraction of the adductors is the greatest obstacle to reduction, nor are they sure that a two weeks' paralysis of these muscles would sufficiently relax a long existing shortening. Moreover, it is rarely difficult to stretch the adductors, manually or mechanically, sufficiently to overcome the obstacle which they present to reduction; and after the head has been replaced, their tightness is rather to be desired as a retentive force. The most serious danger of Robertson's procedure would seem to be the lack of certainty as to return of power in the ligated nerve. In our experience, permanent damage to nerve supply has occasionally

10. Robertson: Surg., Gynec. & Obst., Aug., 1922, p. 230.

been suffered by accidental constrictions, which have been seemingly less complete and severe than that which the author deliberately produces.]

Patella Bipartita.—Blencke [11] reports two cases of supernumerary patellae or double sesamoids. This condition is congenital, but in both his cases a trauma had been sustained, and the findings were supposed to be the result of the accidents. Blencke calls attention to the importance of making roentgenograms of both knees in industrial cases, and to the ruling out of a calcareous deposit in the prepatellar bursa, spontaneous fractures in tabes, etc. Patella bipartita may have its sesamoid at either the upper or the lower end, and there may be two or more fragments, looking at first sight like fractured pieces, but on close inspection found to have smooth, sharp, noncorrugated edges.

Absence of Both Tibiae; Bone Transplants.—Congdon,[12] encountering a case of congenital absence of both tibiae in a 3 year old child who was much improved and made able to walk by bone transplanting operations, has made an interesting observation. In both legs, he discovered a piece of cartilage occupying the space of a portion of the proximal extremity of the tibia. In one leg, he preserved this, and brought it in contact with his grafts. Subsequent roentgenograms showed that ossification was taking place within the cartilage, and a more stable leg was gained on this side than on the other, from which some of this cartilage had been removed.

Os Tibiale.—In eight cases of painful feet, Peltesohn [13] has found an os tibiale over the scaphoid. He was satisfied that this was the cause of the symptoms in pronated feet resulting from irritation of the syndesmosis, from strain of the tibialis posticus tendon. Plaster strapping and foot supports relieved the symptoms. Peltesohn calls attention to the relative frequency of supernumerary bones in cases of painful feet.

Varus Deformity of the Humerus in Microcephalics and Cretins.—Merlini [14] has made a careful study of the humeri in cases of microcephalics and cretins. The normal angle of the humeral head with the shaft is from 140 to 150 degrees. The angle in microcephalics and cretins is from 104 to 126 degrees. He concludes that (1) varus deformity of the humerus occurs in 33 per cent. of cretins and microcephalics; (2) the deformity never reaches an extreme degree, the angle never being more acute than 100 degrees; (3) this diminution in the angle is constant; the other deformities are variable; (4) a frequent if not constant peculiarity of the humeral head lies in the fact that the

11. Blencke: Ztschr. f. orthop. Chir. **42**:291, 1922.
12. Congdon: Northwest Med., July, 1922.
13. Peltesohn: Klin. Wchnschr., April 15, 1922, p. 783.
14. Merlini: Chir. de. Org. di Mov., July, 1922.

articular cartilage, instead of ending at the margins of the head, is continued over the upper part of the anatomic neck and may cover even the greater tuberosity.

RICKETS

Kjerrulf [15] calls attention to the fact that evidence of rickets is common in skeletons of children of the stone age found in Scandinavian countries. The first full description of the disease was given in 1554.

> About 1600, it became very common in England, and spread over Europe almost in epidemic form. The literature on rachitis shows that the necessity for sunlight and fresh air has always been emphasized by physicians in the treatment, but it has taken nearly three hundred years to supply the scientific proof of the reasons why sunlight and fresh air are so indispensible.

Marfan [16] states that when rachitis develops before the child is 4 months old, the first evidences are noted in the skull. It next attacks the ribs, and last the bones of the limbs. In older children, the ribs exhibit the first changes, and the bones of the upper extremity are affected before the bones of the lower extremity. Roentgenography of the wrist is usually very instructive, the bones here being nearly always involved.

Rickets in India.—Hess,[17] in the Cutter lecture at the Harvard Medical School, spoke of the occurrence of rickets in India. In the Nasik district of the Bombay Presidency, where the population is made up of an upper class of well-to-do Hindus, and a lower group of laborers, who are uneducated and poor, the incidence of the disease is most interesting. It occurs almost exclusively among the prosperous class. The dietary of this upper class is far superior to that of the lower class, among whom the disease is almost absent. The mode of life, however, of the first group, enforced by social and religious custom, particularly among the women and children, is largely an indoor existence. Screened from public view, they lack fresh air, sunlight and exercise. We must abandon the idea that rickets is necessarily a disease of poverty. It is to be found in the breast-fed infants of the well-to-do. It attacks chiefly those who lead an indoor existence.

Vitamins and Rickets.—In an editorial in *The Journal of the American Medical Association*,[18] attention is called to the opinion of the Baltimore group of investigators that the power of certain fats to initiate the healing of rickets depends on the presence in them of a substance which is distinct from fat-soluble vitamin A. The facts seem to demonstrate

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15. Kjerrulf: Hygiea, July 16, 1922.

16. Marfan: Nourrison, May, 1922, p. 145.

17. Hess, cited in: Rickets in India, editorial, J. A. M. A. **78**:1966 (June 24) 1922.

18. A Specific Antirachitic Vitamin, editorial, J. A. M. A. **79**:1050 (Sept. 23) 1922.

the presence of a fourth vitamin in addition to the already known A, B and C factors. The specific property of this fourth vitamin, as far as it can be conjectured, would seem to be to regulate the metabolism of bone. Important as is cod liver oil in the treatment of rickets, it is evidently not the exclusive source of antirachitic potency.

Experimental Rickets.—Shipley [19] well sums up the results of experimental studies in rickets: 1. Rickets is a disease of metabolism, usually due to faulty food. 2. It may be produced in rats by certain diets containing an improper balance between calcium and phosphorus, when an uncharacterized substance associated with certain fats is absent or deficiently supplied. 3. There are two kinds of rickets in rats: one, a low calcium type produced by diets relatively low in calcium but containing an approximately normal amount of phosphorus, other things being equal; the other, a low phosphorus type, produced by diets deficient in phosphorus, with a normal or high calcium content. 4. Some uncharacterized organic substance which is present in abundance in cod liver oil enables the organism to compensate for a defective calcium phosphate ratio in food. 5. Exposure to sunlight or to the rays of the quartz lamp (mercury vapor) will give the same result. 6. Either of these factors will induce healing in the bones of rachitic animals. 7. Starvation will also induce healing. These investigations leave a great many questions still unsettled. Some of them we are in a position to answer; others await further study.

Ultraviolet Radiation.—Kramer, Casparis and Howland [20] report five cases in which children showing clinical evidences of rickets, confirmed by roentgen-ray examination, were treated by systematic exposure to the rays of a mercury vapor quartz lamp. Treatment was followed in every instance by healing of the rachitic process in the bones. The inorganic phosphorus concentration of the serum of these children was low (from 2.7 to 3.2 mg.) before the treatment was initiated, but gradually increased to a maximum of 6 mg. with the appearance of calcium deposition in bones. So far as could be judged, healing of the bones occurred at about the same rate after irradiation as after the administration of cod liver oil. The changes in the phosphorus concentration of the serum were identical with those found after treatment with cod liver oil. The pigmentation of the negro child's skin did not seem to interfere with the action of the light rays, colored children requiring no more intensive treatment to bring about healing than did white children.

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19. Shipley: J. Bone & Joint Surg., Oct., 1922, p. 672.

20. Kramer, B.; Casparis, H., and Howland, J.: Ultraviolet Radiation in Rickets, Am. J. Dis. Child. **24**:20 (July) 1922.

Carbon Light Treatment.—Hess and Unger [21] have employed in the treatment of rickets a carbon arc lamp similar to, but smaller than, that employed in the taking of motion pictures. The infants were exposed nude, except for eye coverings or spectacles, at a distance of from 3 to 9 feet, the exposure varying from fifteen minutes at 3 feet, to two hours at 9 feet. There was no tendency to superficial burn and no tanning of the skin (compare Rollier's theory of pigmentation). Eleven cases were so studied by clinical, chemical and roentgenologic examination repeated at least every month. In every instance, there was a rapid subsidence of the signs of rickets, as was evidenced by roentgen-ray examination, frequently after daily exposures for only two weeks. Improvement consisted of calcification of the lower epiphysis of the radius and ulna, which increased progressively as the treatment continued. Accompanying these changes was the constant increase in the inorganic phosphate of the blood, which in some cases had a tendency to fall after roentgenotherapy was discontinued. Animal experiments showed that carbon lamp exposures of three minutes daily at 3 feet were sufficient to bring about this protective effect.

Adolescent Rickets.—Lewin [22] reports five cases of adolescent rickets. It seemed well established from the statements of the patients' families and earlier photographic records that the marked deformities which were exhibited, chiefly genu varum or genu valgum, had developed during the adolescent period, from 14 to 18 years. Some of these patients had had infantile rickets, but no cause was found for the adolescent condition other than a softened condition of the bones, resulting in a gradual and painless development of the gross deformity during the adolescent period. The disease apparently became quiescent, and operative correction of the deformity was successfully accomplished. Four of the five cases occurred in girls.

Corrective Operations on Severe Rachitic Deformities.—Rhode [23] has employed the following technic in operating in cases of extreme rachitic bowing of the legs. His purpose is to obtain as complete correction as possible and insure regeneration. An Esmarch bandage is applied; and the periosteum is carefully dissected back at the seat of operation, being left in close contact with surrounding tissues. A piece of bone sufficiently large to enable a thorough correction of the deformity is subperiosteally resected, and the cavity temporarily packed with gauze soaked in warm saline solution. The resected piece is split by a saw, and the marrow is removed without being destroyed. The

21. Hess, A. F., and Unger, L. J.: Use of Carbon Arc Light in Prevention and Cure of Rickets, J. A. M. A. **78**:1596 (May 27) 1922.

22. Lewin: Surg., Gynec. & Obst., July, 1922.

23. Rhode: Arch.-f. Orth.- u. Unfall.-Chir. **20**:281, 1922.

cortex is then cut into small pieces with a rongeur. The removed marrow surrounded by these cortical chips is placed in the wound cavity, and the periosteal tube and soft tissues and skin are tightly sutured about it. The Esmarch bandage is removed after the wound is closed, and blood fills the empty spaces inside the periosteal tube. The correction is now made, and plaster is applied, and is left on for three weeks, following which a second plaster is employed for four or five weeks. Some weight bearing is allowed with the second cast. The author reports firm union in eight or nine weeks after operation. In one of his cases, a wedge-shaped osteotomy, four months previously, had resulted in a pseudarthrosis. Union in this case, after Rhode's operation, was as prompt as in his other cases.

[ED. NOTE.—This procedure seems to the Editors to be unnecessarily complicated. An attempt is made to overcome difficulties (nonunion and incomplete correction) which, in their experience at least, occur only in exceptional cases, in which the simpler methods of osteoclasis and wedge-shaped osteotomy have been employed.]

SCOLIOSIS

Treatment by Hinged Plasters.—Hanausek [24] has employed a plaster, cut horizontally and firmly united at the back by a hinge, with a horizontal axis placed at the junction of the two curves. The jacket is put on while the patient is in a standing position. The pressure is applied to the deformity of rotation, but no attempt is made to correct the lateral curve. Wooden levers are used daily on each half to rotate the spine. Sometimes the joint is fitted with cogs. Excellent results are shown in photographs taken eight months after the treatment was begun, and the improvement in the lateral curve is said to be as striking as the improvement in rotation. The article needs to be read to be appreciated.

Fencing Position for the Application of Scoliosis Jackets.—Roederer's [25] illustrations also show good results in grave right dorsal, left lumbar curves from the application of jackets in the so-called fencing position. In a right dorsal, left lumbar curve, the subject is seated on the left buttock; the left knee is flexed; the right leg is extended backward, and the left arm forward, so that the right leg, the trunk and the left arm are in a straight line, forming an angle of 45 degrees with the floor. Fenestrae in the cast allow traction from both sides by means of broad bands. This form of application has been employed in thirty-two cases, and in one month the improvement is said to have been infinitely greater than by the Abbott method.

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24. Hanausek: Rev. d'orthop., March, 1922, p. 130.
25. Roederer: Paris méd., July 15, 1922, p. 1003.

Scoliosis Accompanying Chronic Open Pneumothorax.—Gurd,[26] after a long experience with chronic empyema, states that as a rule chronic infected open pneumothorax, if long continued, is followed by scoliosis. Curvature of the spine is toward the healthy side of the chest, and is consequent on pressure exerted on the spine by the contracting ribs of the diseased side. He has obtained correction of the deformity by means of extensive thoracotomy with removal and division of several ribs.

TUBERCULOSIS

Primary Synovial Foci.—Rogers,[27] arguing from clinical observation, takes exception to the evidence presented by those who maintain that tuberculosis of the joints always arises from a focus in the bone, and that the joint structures are involved as a secondary infection. He feels that clinical evidence obtained from early operation on knee joints in which the articular surfaces are found macroscopically undiseased, while tubercle bacilli are found in excised synovial tissue, argues for at least occasional primary synovial membrane infection in tuberculosis, as in other infections. The progress of the disease as studied by the roentgen rays, he believes, suggests this also.

[ED. NOTE.—While Rogers' clinical evidence is important and his views are shared by many surgeons, he must accept the burden of proof. In substance, Nichols and Allison and other laboratory investigators of tuberculosis of the joints say, "Give us a specimen of tuberculosis of the joints in any stage and we will find a bone focus which we believe to be primary to the synovial infection." It is difficult to obtain these specimens, if they exist, for such joints are not excised. Until it is done, however, and capable pathologists find in such joint material only synovial membrane and capsule tuberculosis, and are unable to find any focus in the bone, the discussion must needs go on, and the opinion of Rogers and many other able clinicians lacks positive proof.]

Tuberculosis in Children as a State Problem.—Brown,[28] writing on the care of surgical tuberculosis in children in the commonwealth of Massachusetts, reaches the following conclusions:

1. At present, we have no accurate statistics in this state as to the number of nonpulmonary cases of tuberculosis. This lack is due to the physicians and hospitals of the state failing to report their cases. This can and should be corrected.

2. Surgery in tuberculosis of children is and always should be the exception, and it should always be made only an incident in the general and necessarily prolonged treatment.

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26. Gurd, F. B.: Scoliosis Accompanying Chronic Infected Open Pneumothorax, Arch. Surg. **5**:366 (Sept.) 1922.

27. Rogers: J. Bone & Joint Surg., Oct., 1922, p. 679.

28. Brown: Boston M. & S. J., Sept. 28, 1922, p. 470.

3. In children, operations for spinal immobilization are not advisable, and in adults without the proper postoperative care, do not give the results claimed.

4. In tuberculosis of the spine in both children and adults, the ambulatory and supportive treatment has not given satisfactory results.

5. In children, the ideal treatment is recumbency for at least from two to three years, followed by carefully observed and protected weight-bearing for two years more.

6. In adults, in selected cases, the operation for spinal immobilization may be advisable, if it can be followed by at least six months' recumbency and a year or more of supportive treatment.

7. There are no state or municipal hospitals with an adequate equipment or a personnel which can adequately carry out the proper prolonged treatment of recumbency and heliotherapy.

8. It is the duty of the medical profession of the state to urge the legislature to provide adequate opportunity for the treatment of non-pulmonary as it is provided for pulmonary cases.

Tuberculosis of the Os Calcis.—Frisch,[29] before the Vienna Society of Physicians, presented a patient operated on by Gersuny eighteen years previously for tuberculosis of the os calcis, which had been excised, and the astragalus mobilized, so that its head occupied the place of the heel process of the os calcis. The patient had a normal gait and no subjective symptoms other than the slight discomfort of a flat foot.

Tuberculous Epiphysitis of the Great Trochanter.—Keith [30] reports a case of tuberculosis of the epiphysis of the great trochanter in a boy of 12. The physical and roentgenologic examination suggested a neoplasm. There were enlarged superficial veins, bone destruction, and the shadow of an outlined tumor, at operation shown to be an abscess. There was a history of injury. Keith thinks tuberculosis of the great trochanter more frequent than the literature would suggest.

Tuberculin Treatment of Bone and Joint Tuberculosis.—Graf [31] has administered tuberculin systematically in 100 cases of surgical tuberculosis in the course of heliotherapeutic treatment. His experience would seem to indicate that tuberculin is contraindicated in all such cases unless the process is sharply localized, torpid and obstinately refractory to other methods of treatment. Heliotherapy seems to reduce the tolerance for tuberculin.

A New Plaster Shell for the Treatment of Pott's Disease in Children.—Schwartz [32] emphasizes the importance of inducing the forma-

29. Frisch: München. med. Wchnschr., 1922, No. 14, p. 531.
30. Keith: Am. J. Roentgenol., Sept., 1922.
31. Graf: Deutsch. Ztschr. f. Chir. **171**:57 (June) 1922.
32. Schwartz: J. Bone & Joint Surg., Oct., 1922, p. 789.

tion of compensatory curves and modifying the shape of the spinous processes without separating the surfaces of the tuberculous vertebrae. The ordinary shell, hinged at the apex of the kyphos, was found by roentgenologic studies to be often applying the fulcrum unmechanically and incorrectly at different levels of the spine. In the frame or shell which he has devised, the head and thighs are supported, as well as the spine. An adjustable fulcrum is arranged as broad as the diseased area. At the level of the seventh dorsal vertebra, the fulcrum is placed opposite the apex of the kyphos. In lesions above the seventh dorsal vertebra, the fulcrum should be placed slightly below the apex of the kyphos. Approximately one quarter inch should be subtracted for each vertebra above this level. In lesions below the eighth dorsal vertebra, the fulcrum should be placed slightly above the apex of the kyphos, adding approximately one quarter inch for each vertebra below this level. Employment of the shell, mounted on uprights, permits the taking of lateral roentgenograms as controls of mechanical efficiency, while the patient is resting in it.

POLIOMYELITIS AND SPASTIC PARALYSIS

Communicability and Serum Treatment of Poliomyelitis.—Amoss [33] summarizes the work from the Rockefeller Laboratory thus:

Distribution of the virus in the host: The virus has not been found in either the cerebrospinal fluid nor the blood of human subjects, but has been found in both in monkeys. The virus has been found in the nasal washings of both human and monkey cases. The definite time of disappearance has not been determined. There is doubt concerning the virus in the stools.

Route of Infection: It appears that the virus is transferred by chance either directly or indirectly from the nasal mucous membrane of the host to the nasal mucosa of the susceptible new host. It is propagated in the central nervous tissues of the host, but does not survive external conditions of mediation.

Defense Mechanism: The nasal washings of healthy adults, but not of children, neutralize the virus. In monkeys large doses intravenously rarely cause infection. It has been observed that a positive Schick test in poliomyelitic children is present twice as often as the expected occurrence in children of the same age in the general population.

Carriers: The virus seems to have a greater chance of surviving in the nasal secretions of a carrier than in the recovered case. Healthy carriers have been demonstrated.

Communicability: The stage of communicability is from one week before to two weeks after the onset.

Prevention: In Vermont the patient and the intimate contacts are quarantined for three weeks.

Treatment: Since the lesion is tucked away in the most remote and inaccessible parts of the body, treatment is a difficult art. Serum from recently recovered cases is recommended, as it is believed to have a greater antibody content. The serum for intraspinal injection must be free from particles and

33. Amoss: New York State J. Med., June, 1922, p. 256.

hemoglobin. The results from serum treatment seem to show definitely when analyzed that the disease is arrested in cases treated within forty-eight hours after the onset with more than 50 c.c. of the serum.

Poliomyelitis During Influenza Epidemics.—Hiller [34] reports ten cases of affections of the spinal cord during a recent influenza epidemic in Dresden. In six of the cases, the clinical picture was that of an acute anterior poliomyelitis. In three, it resembled an acute diffuse myelitis, and in one there was a sudden paralysis of the bladder. The latter patient recovered completely. The paralysis in the others slowly retrogressed, but traces of it remained. Hiller's study of the records shows an epidemic appearance of disease of the central nervous system in the great influenza epidemics of 1712 and 1889-1890.

Diagnosis, Prognosis, and Early Treatment of Poliomyelitis.—Lovett's [35] long experience makes the article of great value, especially in a disease in which every case is an individual anatomic study. The special points he makes are: 1. Diagnosis should be made on a basis of physical examination rather than on the history. 2. Tenderness is a guide of great importance in diagnosis, prognosis, and treatment. 3. Early treatment consists of rest. 4. In the convalescent stage, muscle fatigue is our chief danger, and muscle reeducation our chief reliance. 5. Deformities prevent proper function and favor muscular deterioration.

Treatment of Poliomyelitis in Children.—Girdlestone [36] describes the principles of treatment laid down by Sir Robert Jones. At the onset, the child is wrapped in cotton wool and kept absolutely at rest for six weeks or more, plaster or retentive splints being applied over the wool. Next comes the stage of gentle massage. In the second stage, the principles carried out are: (1) posturization of the trunk and limbs; (2) correction of deformities; (3) resumption of function of the ganglion cell and the affected part by slow stimulation of light massage, reeducation, guarded active movements and locomotion without overstrain. Electricity is considered of doubtful value.

Acute Poliomyelitis in an Adult.—Winther [37] reports a carefully studied case of poliomyelitis of the Landry type, a sporadic one, running a very acute course, and fatal in eighteen hours. Paralysis occurred as a result of the involvement of the anterior horn motor cells of the respiratory muscles: first, the diaphragm (phrenic nerve), then the intercostal muscles, and finally the auxiliary respiratory muscles. A detailed account is given of the histologic examination of the cord in the region involved.

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34. Hiller: Deutsch. Arch. f. klin. Med., May 5, 1922, p. 143.

35. Lovett, R. W.: Diagnosis, Prognosis and Early Treatment of Poliomyelitis, J. A. M. A. **78**:607 (May 27) 1922.

36. Girdlestone: Brit. M. J., Sept. 20, 1922, p. 596.

37. Winther: Hospitalstidende, July 5, 1922.

Accessory Muscle Movements in Deltoid Paralysis.—Pollock [38] discusses, on the basis of four observed cases, the supplementary motility in abduction in complete paralysis of the deltoid. He cites Bunts' report of nineteen cases of paralysis of the circumflex nerve, in seven of which function was recovered. In four of these, there was no deltoid power. Pollock points out the importance of the long head of the biceps and of the supraspinatus and infraspinatus muscles. In combined action with the coracobrachialis, these muscles may be able to abduct the arm 65 degrees. When the head of the bone is firmly fixed in the glenoid fossa by the subscapularis and the pectoralis major, the serratus magnus, trapezius, rhomboids and levator anguli scapulae may bring about fair abduction.

Static Relations Between Foot and Knee in Paralyzed Legs.—In cases of total paralysis of the foot associated with paralysis of the quadriceps, Putti [39] has obtained a successful blocking of dorsal flexion of the foot at an angle of 110 degrees, by inserting a graft from the shaft of the tibia into the anterior part of the astragalus. The top of the graft is so placed as to impinge on the anterior margin of the tibia. In cases of instability of the subastragalar joints, he adds an arthrodesis between the astragalus and os calcis, and between the astragalus and scaphoid. He has performed the operation in six cases, all due to poliomyelitis, in children between 7 and 16 years of age. The period of observation following the operation varied from two years and four months to six months. The results to date of last observations, both clinically and from the roentgenograms, were gratifying.

[ED. NOTE.—Discomfort from impingement following fractures in this region is common, and we see no reason why such discomfort should not obtain if the blocking was deliberately produced as is proposed in this operation.]

Spastic Conditions: Intraperineural Neurotomy.—Nutter [40] considers that intraperineural neurotomy should be performed only in those cases that can be under the surgeon's care for several years, and in which spasm of the muscles is the chief cause of loss of function. This spasm may be limited to a few muscles and yet standing and walking be practically impossible, rendering the patient incapable of mingling with other children. Nutter outlines the technic and after-treatment which he employs.

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38. Pollock, L. J.: Accessory Muscle Movements in Deltoid Paralysis, J. A. M. A. **79**:526 (Aug. 12) 1922.

39. Putti: Chir. de. Org. di Mov., April, 1922.

40. Nutter: J. Bone & Joint Surg., July, 1922, p. 453.

ARTHRITIS

Types of Chronic Arthritis.—Swaim [41] described three types of arthritis which he believes are distinct in their etiology and pathology, and in their clinical and roentgenologic manifestations. He employs Goldthwait's nomenclature, calling them infectious, atrophic and hypertrophic. The response to treatment differs in the different types, and he considers clinical data of value only when these types are studied as separate pathologic entities. The closest association between the internist and the orthopedic surgeon should exist in treatment. It should be the function of the internist to discover the cause and to arrest activity; the function of the orthopedic surgeon, to prevent and correct deformity and to preserve function. Normal body function and normal body mechanics are interdependent. Restoration of normal body mechanics may turn the scale favorably.

The Second Great Type of Chronic Arthritis.—Ely [42] considers that sufficient evidence has been accumulated to indicate that the second great type of chronic arthritis is probably caused by a protozoon which almost invariably gains access to the system through the diseased bone at the roots of decayed teeth. Its prime characteristic is an aseptic necrosis in the marrow in the immediate vicinity of the joint. It is recognized roentgenologically by the presence of spurs or lipping at the lines of attachment of the capsule. It occurs about twice as often in men as in women. It is a disease of middle or late adult life, never occurring before the third decade. Up to the present time, the best results in the treatment have been the removal of the focus in the alveolar processes of the jaws.

Amebiasis of Bones.—Kofoid and Swezy [43] examined material from the excised head of a femur, in the immediate neighborhood of the necrotic areas, close to the eburnated surface of the joint. The material was received from Ely's clinic, he having obtained it from a case belonging to his second great type of arthritis. Kofoid and Swezy found ameboid cells which they interpret as *Endameba dysenteriae* in the specimen. The question arises as to whether the ameba of the bone is that of the bowel (*Endameba dysenteriae*), or the ameba of the teeth and tonsils (*Endameba gingivalis*). From their critical morphologic studies, they incline to the view that the ameba of the bone is *Ameba dysenteriae* rather than *Ameba gingivalis*. They state that, in their experience, coexistence of infection by *Ameba dysenteriae* and chronic arthritis has been frequent. They believe that only a tentative conclusion can as yet be reached.

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41. Swaim: J. Bone & Joint Surg., July, 1922, p. 426.

42. Ely: California State J. Med., Oct., 1922, p. 329.

43. Kofoid, C. A., and Swezy, Olive: Amebiasis of Bones, J. A. M. A. **78:** 1602 (May 27) 1922.

[Ed. Note.—Swaim's classification has the advantage of being a working classification, but the disadvantage of being a mixed etiologic and pathologic one. Ely's classification of Type I and Type II has at least the merit of simplicity, though it cannot be said to be descriptive. We know that there are forms of chronic arthritis in which infection, local or remote, plays the most important etiologic rôle. These are so many and so varied that our present knowledge perhaps forbids an accurate pathologic classification. Nichols and Richardson, finding that in this type the synovial membrane changes consisting in villous formation came early, proposed the name of proliferative arthritis; and noting that ankylosis was the usual end-result, also spoke of the type as the ankylosing type. This type corresponds to Ely's Type I. Nichols and Richardson recognized that another distinct type existed, in which the early joint changes were represented by a fibrillation and degeneration of the cartilage, and that the terminal changes rarely resulted in bony ankylosis. They, therefore, speak of a degenerative or non-ankylosing type, which corresponds to Ely's Type II and Swaim's hypertrophic type. They could find no evidence clinically or pathologically of an infection in this type, agreeing with Swaim and disagreeing with Ely's latest opinion. Whether there is, as Swaim holds, a third distinct type, which Goldthwait, Swaim, and others, notably McCrae, call the atrophic type, the Editors are not certain, though undoubtedly there are many cases in which atrophic areas and bone destruction appear at the joint line very early, associated with vasomotor changes.

Such changes may be produced with certainty in the bones of guinea-pigs fed on a scorbutic diet, as Dr. Percy Howe at the Forsyth Dental Infirmary has so well shown. These joints go on to ankylosis, but whether these changes are purely metabolic or chemical, or whether they are the direct result of bacterial infection made possible by the lowered joint and bone tissue resistance to bacterial body habitants, harmless under normal conditions, remains to be demonstrated. Certain it is, as Swaim says, that the internist and the orthopedic surgeon must labor together in the study of perhaps the most important chronic disease that exists, and must attack the problem from both the biochemical and the bacterial approaches, and from the vantage ground of improvement in body mechanics and restoration of function. We trust that Ely, working perhaps with such an eminent protozoologist as Kofoid, will continue his investigations as to the etiologic importance of *Ameba dysenteriae* or *Ameba gingivalis*.]

Acute Articular Typhoid.—Thiroloix and Harmelin [44] report a case of acute multiple arthritis which developed shortly after injury to the

44. Thiroloix and Harmelin: Bull. et mem. Soc. méd. d. hôp. de Paris, July 7, 1922, p. 1054.

feet, in a young man. Multiple hemorrhages occurred, and typhoid bacilli were discovered in the blood at the end of the third week. Salicylates proved of no avail, and the exsanguinated patient died in two months.

Spondylitis Due to Leukemia.—Melchior [45] describes a case of a boy of 11, who complained of rather sudden onset of pain in the lumbar spine. At examination two months later, rigidity and tenderness were found, and a diagnosis of tuberculous caries was made. An Albee operation was performed, but an infection occurred and the graft was removed. The blood showed a leukemia, and at necropsy, two months later, the fifth lumbar vertebra was found destroyed by lymphatic cells. A lymphatic focus was also present in the femur. One previous similar case was reported in the *Lancet* in 1914, page 173.

The Effect of Sodium Salicylate on the Development of Arthritis in Rabbits.—Boots and Swift [46] have carried out a series of experiments to determine whether sodium salicylate administered to rabbits by stomach tube in doses comparable to those given to patients with acute rheumatic fever would prevent arthritis in animals inoculated with cultures of *Streptococcus viridans.* Fifty rabbits were studied. Twenty-five were inoculated and twenty-five were used as controls. Twenty-three developed arthritis, and the authors consider that this proves that salicylates do not prevent arthritis. The animals receiving the salicylates, however, seemed from an analysis of the joint fluids to have a less severe arthritis than did the controls.

Sulphur Treatment of Chronic Arthritis.—Weskott's [47] experiences with the administration of sulphur in the treatment of chronic arthritis have not been so favorable as he had expected them to be from the reports of Meyer-Bisch and the French authors. He encountered severe general reactions, such as vomiting and tremulousness, which persisted for several hours. In many cases, there was little if any relief from pain in the affected joints.

Tonsillectomies for Rheumatism in Adults.—Hastings [48] has reviewed critically the results of 130 tonsillectomies performed on adults between 19 and 65 for the purpose of relieving rheumatic symptoms of pain and stiffness in the joints, requiring in some cases crutches and apparatus. There were no cases of definite, well established "rheumatoid arthritis," and none of the patients were bedridden. Previous throat trouble had

45. Melchior: Zentralbl. f. Chir., Nov. 25, 1922, p. 1737.

46. Boots, R. H., and Swift, H. F.: The Effects of Sodium Salicylate on the Development of Arthritis in Rabbits Inoculated with Streptococcus Viridans, abstr. J. A. M. A. **78**:1922 (June 17) 1922.

47. Weskott: München. Med. Wchnschr., 1922, No. 18, p. 674.

48. Hastings: Ann. Otol., Rhinol. & Laryngol., March, 1922.

existed in about 66 per cent. of the cases. Sixty-nine cases were classed as mild chronic arthritis; twenty-eight, as moderate, and four, as severe; three, as acute arthritis; six, as neuritis, and twenty as myalgia.

Of these 130 patients, Hastings considered from observation and reports that forty, or about 30 per cent., were cured, fourteen, or about 9 per cent., were greatly improved and twenty-four, or 19 per cent., not improved. In twenty-nine cases, the results could not be traced.

Absorption from Joints.—S. Draganesco and A. L. Draganesco [49] have studied the absorptive power of joints by injecting various drugs, such as potassium iodid and methylene blue, into diseased joints, and examining the urine thereafter. They also injected the drugs subcutaneously and examined the joint fluids. They conclude that joints possess considerable absorbing power for fluids injected directly into them but not for drugs injected elsewhere in the body subcutaneously.

NEOPLASMS

Pathologic Fractures.—Codman,[50] in an interesting short paper read before the Clinical Congress, American College of Surgeons, makes the following chief points regarding pathologic fractures: 1. Fracture is the symptom of onset in a central lesion suggests bone cyst, and if the patient is 15 years of age or younger, it may be looked on as almost pathognomonic (Bloodgood). 2. Pathologic fractures are usually transverse, with little tendency to displacement. They are liable to be bendings, bowings, bucklings, telescopings, and impactions. 3. A safe surgical nomenclature of bone tumors is the following: (*a*) osteitis fibrosa, which includes ordinary bone cysts and the hemorrhagic osteomyelitis of Barrie; (*b*) Bloodgood's giant cell tumor, called myeloma in Canada and Europe; which may prove malignant; (*c*) giant cell sarcoma—an epulis-like tumor with "foreign body giant cells"—which eventually proves malignant; (*d*) true osteogenetic sarcoma, either central or periosteal; (*e*) metastatic new growths, cancer, hypernephroma, etc., (*f*) multiple myeloma, with or without albuminosuria; (*g*) tumors of tissues which normally occur in bone as well as in other parts of the body, such as nerves, vessels and fat, causing bone destruction and proliferation secondarily (fortunately rare); (*h*) tumors too common and well understood to be included, e. g., chondroma, osteoma, and lesions of syphilis and tuberculosis; (*i*) tumors too rare and little understood to be taken into practical consideration, e. g., the tumors of the specific marrow cells. 4. Fractures due to benign bone tumors and other pathologic conditions, excluding new growths, usually unite.

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49. Draganesco, S., and Draganesco, A. L.: Presse méd., Aug. 30, 1922, p. 1368.

50. Codman: Surg., Gynec. & Obst., May, 1922.

Those due to carcinoma and other metastases rarely unite. 5. Judging from the material accumulated by the Registry of Bone Sarcoma, it should be a general rule to employ splints in all cases of tumors of the long bones as soon as they are observed, particularly those of the humerus and femur, which are not supported by companion bones. Pathologic fracture occurred in 14 per cent. of 200 supposed cases of sarcomas of the long bones. Of nineteen humeral cases, it occurred in 47 per cent.; of the 102 femur cases, in 15 per cent.; and of the seventy-nine cases of the four self-splinting bones, in only 5 per cent. 6. As regards fracture as a cause of sarcoma, exuberant callus resembles true sarcoma and perhaps the cause will be found to be a negative rather than a positive one, such as the removal of an inhibition in the process of repair. On this theory, malignancy would be an unbridled repair process.

Hemorrhagic Osteomyelitis.—Barrie [51] has personally observed forty-two cases of what he terms hemorrhagic osteomyelitis. The age incidence was from 18 months to 63 years. Seventy-five per cent. of these solitary lesions had, according to Barrie, a traumatic etiology. Bacteria were never isolated in any of the removed tissue. The lesion occurred in the lower extremity three times as often as in the upper extremity, in the femur most frequently, and next in the tibia. Pressure always revealed tenderness and elicited pain. Signs of inflammation were lacking. The dominant pathologic picture is that of highly vascular granulation tissue filling a bone cavity. There may be localized fibrous metaplastic areas and numerous scattered hyaline bodies in and around the hemorrhagic mass. Scavenger giant cells may be many or few, but true tumor giant cells are never found in these lesions. A preoperative diagnosis without a roentgenogram is impossible. There is usually in the early lesions a clear-cut, rounded or oval spot on the cortex. Expansion of the bone is observed without breaking through the periosteum. Any chronic bone lesion which has broken through the periosteum should be regarded with suspicion as to malignancy unless the lesion is known to be definitely suppurative. The treatment consists of removing all exuberant granulations and thoroughly curetting the bone cavity wall. Swabbing out with tincture of iodin may stimulate normal bone production. If hemorrhage is severe, packing for forty-eight hours may be necessary. The healing of large cavities is promoted by filling with bone graft shavings or chips.

Sarcoma of Bone.—Gibbon [52] describes a lesion in the lower end of the femur which, from the roentgenogram, was diagnosed as a new growth and at operation was thought to be a giant cell tumor. Micro-

51. Barrie: J. Bone & Joint Surg., Oct., 1922, p. 653.
52. Gibbon: J. Bone & Joint Surg., July, 1922, p. 512.

scopically, it proved to be a large round cell sarcoma with a few giant cells. The growth was removed as completely as possible, amputation being refused. Recurrence in three weeks necessitated a second operation, and four weeks later amputation was necessary. The case is of interest as occurring in a 16 year old boy, and because both roentgenographically and in gross specimen it closely resembled a giant cell growth. The author urges the importance of accurate microscopic diagnosis.

Total Removal of the Scapula for Primary Giant Cell Sarcoma.—Davis [53] reports the occurrence of a tumor of the scapula necessitating total excision, in a patient, aged 24. The tumor proved on pathologic examination to be a large giant cell sarcoma. Ten weeks following the operation, the patient reported by letter that she was doing general house work without difficulty and was able to make beds, wash dishes, sweep, dust and iron, and comb her own hair. The operation was performed as follows: An incision was made over the tip of the coracoid, extending outward along the coracobrachialis. The tip of the coracoid was exposed, and the attachments of the pectoralis minor, coracobrachialis and short head of the biceps were divided. The subscapular artery and vein were freed and ligated. An incision was then made from the superior angle of the scapula along the vertebral border to the lower angle, and this was connected with another incision extending from the tip of the acromion along the spine of the scapula. The muscles attached to the scapula were divided in the following order: trapezius, the rhomboids, latissimus dorsi and teres major and minor. The serratus magnus was cut just as it passed under the external border of the subscapularis. The lower angle of the scapula was then elevated and the remaining muscles, consisting of the levator anguli scapuli, omohyoid and deltoid, were cut as they came into view. The descending branches of the transverse colic and the transverse scapular arteries were cut and tied. The acromion was then disarticulated from the clavicle, and the shoulder joint was opened posteriorly. The long head of the biceps and the triceps attachment were divided, and the glenoid process was removed from its connection with the humerus. A running suture was employed to gather together the mass of muscles and form a muscular pocket about the head of the humerus. The wound was closed with drainage, and the arm was dressed in slight abduction by means of a thick axillary pad.

Prevalence of Bone Sarcoma in Massachusetts.—Codman [54] makes a further report of the progress of the Registry of Bone Sarcoma. In

53. Davis: Surg., Gynec. & Obst., June, 1922.
54. Codman: Boston M. & S. J., Oct. 12, 1922, p. 543.

a previous article, seventy-one living persons presenting possible cases had been located in Massachusetts. Further investigation has reduced this number to nine. If giant cell tumors should be included, there would be at least twenty-two more. Four practical points have been thus far brought out: 1. The diagnosis should be made with great caution. 2. Bloodgood's claim as to the benign character of giant cell tumors is confirmed by the experience in Massachusetts. 3. True osteogenetic sarcoma is almost always fatal, the rare exceptions occurring in cases in which amputation is performed. 4. Since only nine persons having bone sarcoma (excluding the giant cell tumors) can be located in a population of approximately 4,000,000, there are probably only 225 in the United States. Codman urges the importance of registering persons who refuse operation but are treated with roentgen rays, radium, Coley's serum, etc.

Roentgen-Ray Diagnosis of Tumors of Long Bones.—Nichols,[55] in an endeavor to simplify the roentgenologic diagnosis of bone tumors, suggests a differential method based on the observation of four fundamental points: (1) their origin, whether medullary or cortical; (2) whether or not they are characterized by bone production, by bone destruction, or both; (3) the resultant condition of the cortex, whether expanded or destroyed, and (4) whether the growth is invasive or noninvasive. His classification of tumors is presented in the accompanying table:

CLASSIFICATION OF TUMORS

1. Origin		
a. Medullary	Sarcoma, Carcinoma, Hypernephroma, Myeloma	Fibroma, Bone cyst, Enchondroma, Giant cell sarcoma
b. Cortical	Periosteal sarcoma, Osteosarcoma, Osteoma, Enchondroma	Ossifying hematoma, Myositis ossificans, Bone cyst (rare), Exostoses
2. Bone Production	Periosteal sarcoma, Osteosarcoma, Osteoma, Exostoses	Ossifying hematoma, Myositis ossificans, Enchondroma, Bone cyst after fracture
3. Condition of Cortex		
a. Expanded	Enchondroma, Bone cyst	Giant cell sarcoma, Fibroma
b. Destroyed	Sarcoma, Carcinoma	Hypernephroma, Myeloma, Periosteal sarcoma
4. Invasion		
a. Invasive: All malignant tumors		
b. Noninvasive: All benign tumors		

55. Nichols: Surg., Gynec. & Obst., Sept., 1922, p. 301.

Tumors of Bone.—Bloodgood [56] suggests the following working rule for the diagnosis and treatment of tumors of bone. If a patient is under 15 years, we can exclude sarcoma, and the diagnosis rests between the common bone cyst, the less frequent giant cell tumor and the rare chondromyxoma. If the patient is over 15, sarcoma cannot be excluded. The most common central lesion of bone is the benign bone cyst. In the majority of cases, they recover without any treatment. The second is the giant cell tumor, which predominates in patients over 15. Myxoma may occur at any age from birth to death. Myxoma must be constantly borne in mind, and is the most difficult of all bone tumors to eradicate locally.

The location of the tumor is also important: whether in the end of the bone, in the joint, or in the middle of the bone near the nutrient foramen; the last being the favorite site of the metastasizing carcinoma. Malignant tumors do not cross a joint, so that a lesion involving both sides of the joint is always a benign process. Whether the tumor is single or multiple, is also important, as is also the age of the patient.

[Ed. Note.—Differential diagnosis of bone tumors is far from certain by any known method, particularly roentgenoscopy. This is altogether natural when we remember that even with the gross specimen before him and the slide under the microscope, the pathologist is often uncertain as to its character. Often there is such widespread involvement of the bone that it is impossible to determine the point of origin or the invasion. While in sympathy with any effort to systematize the diagnosis, we are under the impression that the one point of importance is whether the growth is benign or malignant, and that the best way to determine this is by immediate exploration and pathologic examination. The relation of the region to be examined is also of importance, and occasionally the surgeon well trained in gross appearances in malignant disease must be governed by his finding quite as much as by the report of the pathologist, made necessarily somewhat incompletely at the time of operation.]

Fibrous Tumor of the Foot.—Thorek [57] reports two unusual cases of large benign fibrous tumors of the foot, one originating near the internal malleolus and the other in the plantar fascia. Both were widely excised, and the skin defect was closed at a later stage by whole thickness grafts. A review of the literature reveals the extreme rarity of this type of growth, the majority of such tumors being malignant.

Carcinoma of the Vertebrae.—Sicard [58] calls attention to the differential diagnosis between cancer of the spine and tuberculous caries by

56. Bloodgood: Northwest Med., Sept., 1922.
57. Thorek: Ann. Surg., July, 1922.
58. Sicard: Bull. et mem. Soc. méd. d. hôp. de Paris, June 16, 1922, p. 920.

means of roentgenograms. The condition of the intervertebral disk he considers indicative. In Pott's disease, the disk is early affected or destroyed, while in carcinoma it is comparatively intact and the body is chiefly affected. He considers that the explanation of this lack of disk involvement in cancer may be found in the poor blood supply of the disk, while the metastasizing cells are planted in the bodies of the vertebrae by their richer circulation.

Bone Metastasis from Primary Carcinoma of the Urinary Bladder.—Wells,[59] in five cases of cancer of the urinary bladder, in which necropsy was performed, found only one with bone metastasis. The main tumor was in the sternum, in several of the ribs and in the ilium. There had been no symptoms referable to the bladder in this case.

OSTEOMYELITIS

Regeneration of Shaft of Humerus Following Acute Osteomyelitis.—Nifong[60] reports a case of complete regeneration of the shaft of the humerus in a child of 12, in which there had been destruction en masse, so that the sequestrum represented all the shaft except the articular ends. The periosteum with barely palpable flakes of bone represented the involucrum. No transplant was employed, but the periosteum was sutured together. Complete reformation of the humerus occurred after twenty-three months, and motion was only a little short of normal.

An Analysis of One Hundred and Sixty Cases of Osteomyelitis, with End-Results.—Speed,[61] for purposes of report and study, divides osteomyelitis into four types: acute localized, chronic localized, acute diffuse and chronic diffuse. Twenty-nine of his 160 cases were localized and 131 diffuse. Eighty per cent. of the diffuse type occurred before the age of 15. Those of localized type were cured by thorough curettage and removal of the localized disease. In the acute diffuse type, twenty-one cases, there was a definite history of trauma in 55 per cent. In this group, there were four cases showing multiple lesions. A conservative, trap door, simple drainage of the bone was established as soon as the diagnosis was made. For purposes of this report, the author considers a patient cured when no symptoms have been evident for at least a year. There were nine apparently complete cures, five incomplete cures and seven deaths. The high mortality is accounted for by the fact that several of the patients were in a desperate condition when brought to the hospital. There were 110 cases of the chronic diffuse type. The average number of operations performed in each case was three. Ten per cent. of the lesions had been healed for vary-

59. Wells: J. Urol., May, 1922.
60. Nifong: Missouri State M. A. J., May, 1922.
61. Speed: Southern M. J., Sept., 1922, p. 721.

ing periods of from six months to fifteen years, at various times, before the recurrence which brought them to the hospital. Of the sixty-four persons operated on, one died of sepsis, 14 per cent. were still undergoing drainage and 84 per cent. were cured, or at least had remained without symptoms for at least one year. The records of this series do not favor complete subperiosteal resection of the tibia, radius or ulna, even when practically the entire shaft is involved. Any loss of continuity of bone is to be avoided. Defects of only a half inch cause a marked prolongation of the period of disability. Simple fractures through the involcrum have all united much like normal bone.

Chronic Bone Abscess.—Brickner,[62] proceeding on the theory that in chronic bone abscess of afebrile character the infective organism has either been entirely destroyed or at least greatly attenuated in virulence, advocates the treatment of such cases by conservative rather than by radical surgical procedures. His procedure consists in exposing the bone through a small incision and driving a drill, one-fourth inch in diameter, through the cortex into the pus cavity. With the escape of pus, the drill is removed and nothing else is introduced into the bone. A culture is made to determine what, if any, living organisms are present, and a smear is also made and examined at once. If, instead of none or a few bacteria, many are found, the procedure may be altered. A small drain of folded rubber dam is laid in the soft parts, down to but not into the bone opening, and the wound is closed. The author has used this method in selected cases for several years, with relief of pain and without recurrence of symptoms.

[ED. NOTE.—We have been impressed with the fact, as pointed out in previous reports, that there is need for greater conservatism in the treatment of chronic osteomyelitis; and this is substantiated by Brickner's observations. At the same time, it must be admitted that no conclusions as to the end-results of treatment in cases of osteomyelitis are possible until the lapse of many years, and opinions as to the value of this particular method should not be formed until the case reports and end-results are published.]

BONE, JOINT AND TENDON SURGERY

Beef Bone Pegs.—Tarnowsky[63] has employed a beef-bone intramedullary peg in fifteen cases of recent fractures, for internal fixation, and in two cases of ununited fracture of the neck of the femur; with success in all but one case. Union occurred and the bone was well tolerated. He considers the fate of these heterogenous grafts to be dependent on the following considerations: 1. With the proliferation of new

62. Brickner: J. Gynec. & Obst., July, 1922.
63. Tarnowsky: Surg., Gynec. & Obst., Sept., 1922, p. 342.

cells from the living proximal and distal ends of the fractured bone keeping pace with absorption of dead cells from the graft, complete disappearance of the latter coincides with complete union of the fracture. 2. With hyperactive new cell formation and sluggish absorption, the graft constitutes a sequestrum which either eburnates and remains in a state of quiescence, or may act as an irritant foreign body, with sinus formation. 3. With hyperactive bone absorption and sluggish new bone formation, the graft disappears by combined extrusion and absorption before union takes place.

Girode,[64] on the other hand, in Delbet's clinic, has had a most unfavorable experience with beef bone implants in fifteen cases of pseudarthrosis or fracture of the neck of the femur. The bone was obtained from the tibia or ulna of freshly slaughtered cattle and preserved in alcohol. Delbet has abandoned the method.

Beef Bone in Stabilizing Operations on the Spine.—Brown[65] has employed boiled beef bone, cut from the rib, carefully sterilized by boiling, and inserted after the manner of an Albee graft into the split spinous processes. He reports results in thirty-three adult patients and one child. Twenty-nine were operated on for tuberculosis of the spine, four for old fractures, and one, an adult, for an unstable spine following poliomyelitis. It was demonstrated that the beef bone was tolerated by the body for as long as four and one-half years without any sign of irritation or inflammation and only slight indications of absorption. The end-results in this series and in experimental work on dogs leads the author to feel that immobilization of the spine can be more surely accomplished by fusion of the laminae and articular facets than by means of a beef-bone splint inserted between the spinous processes, even if the laminae and articular facets are fused on one side, in addition. In the cases of tuberculosis of the spine in adults, a careful investigation revealed the fact that half of the cases had more than one focus of tuberculous infection, and in six there were two separate foci in the spine.

[Ed. Note.—This rather extensive and careful work of Brown is of value and seems to the editors to indicate that surgeons will probably be disappointed if they depend on boiled beef bone to act more than temporarily as a nonirritating internal splint. In several of Brown's cases, it was possible to reexamine the splint when a second operation was performed for a later developing second focus, and in one case in which the graft was broken from excessive lifting. Although the beef bone was found to have been perfectly tolerated, it showed little sign of actual fusion with the spinous processes and was in places

64. Girode: Rev. de chir., **60**:60, 1922.
65. Brown: J. Bone & Joint Surg., Oct., 1922, p. 711.

lying free in a tube of bone, the tips of the spinous process having united over it, but not with it. In other places, absorption was slowly taking place. In the light of these studies, it seems to us that the procedure which Kleinberg [66] has employed in stabilizing scoliotic spines is not likely to accomplish its purpose. This consists, in addition to a fusion operation, of implanting a long beef bone splint on the concave side of the curve into two spinous processes above and below, which have no intimate contact with denuded bone over a considerable span. We doubt the permanent efficiency of this supporting strut.]

Transplantation of the Fibula.—MacAusland and Sargent [67] report successful cases of transplantation of the fibula to replace the loss of the entire shaft of the tibia following osteomyelitis. The upper and lower ends of the bone were transplanted at different periods in order to preserve the blood supply. Hypertrophy of the fibula resulted, with normal function of the ankle joint and no instability.

Para-Articular Arthrodesis of the Hip Joint.—Kappis [68] has immobilized the hip joint in fourteen cases of old tuberculous coxitis by implanting a heavy bone and periosteal graft between the trochanter and the iliac crest. He considers that the results justify the extensive use of this method.

Nové-Josserand [69] also, in discussing the indications for the different methods of surgical fixation of a joint, comments on Maragliano's [70] success with a long bone graft similar to the graft employed by Kappis. These grafts retained their vitality, although contacted only at their two ends. He points out that this procedure runs no risk of lighting up the old tuberculous process. The results he considers most satisfactory. He prophesies a brilliant future for the method.

[ED. NOTE.—This procedure would in no way correct existing deformity, which is often one of the main reasons for attempting an arthrodesis. We have rarely if ever met a recurrence of the old disease after a well-executed arthrodesis, and we should also doubt the permanence in many cases of this graft, contacted, as it must be, with such a thin bone as the ilium.]

Resection of the Knee in Children.—Nussbaum [71] has reviewed the excellent records of Garré in regard to the end-results of resection of the knee joint in children. Shortening was the most important finding, the amount bearing some relation to the age of the child, as was

66. Kleinberg: New York M. J., July 19, 1922, p. 93.
67. MacAusland and Sargent: Ann. Surg., July, 1922.
68. Kappis: Deutsch. Ztschr. f. Chir., March, 1922, p. 316.
69. Nové-Josserand: Paris méd., July 15, 1922.
70. Maragliano: Paris méd., May 20, 1922, p. 1579.
71. Nussbaum: Beitr. z. klin. Chir. **126**:662, 1922.

to be expected. When the epiphyseal lines were left undisturbed, the shortening was less marked, but in many cases even thus conservatively treated, it was considerable. Another important fact was the participation of the foot in the disturbance of development, the cavus deformity which ensued being noticeable. A subsequent flexion of the knee developed in several cases. The course of the disease was undoubtedly shortened in many cases, and Nussbaum considers that there may be social indications for the operation.

A New Approach for Ankylosis of the Jaw.—Beckenheimer [72] proposes a new approach to the temporomaxillary articulation for an arthroplastic operation in cases of ankylosis. A vertical incision is made just behind the auricle, avoiding above the vessels to the outer portion of the ear and not extending low enough to reach the facial nerve. The auricle is turned forward, and the external meatus divided. He states that the exposure of the joint is excellent, and the method has the great advantage of safety as far as the facial nerve and the parotid gland are concerned. The condyle is resected and a fascial flap interposed. The auricle is carefully stitched in place and a drain placed in the meatus. The scar is unnoticeable.

Arthroplasty.—Putti [73] has on record 113 cases of operative mobilization of stiffened joints: forty of the knee, thirty-eight of the elbow, seventeen of the hip and ten of the jaws. He considers that the greatest advance which has been made in the operation is the interposition of the free fascial transplant. His general principles embody a free exposure of the joint, cutting as few as possible of the structures which induce motion and are essential to weightbearing. He covers both ends of the bone with free fascial flaps. He never drains the new joint and lays great stress on traction on the distal segment. This he maintains for a month. Passive movements carried out by the patient himself, by means of a simple suspension apparatus, are begun on the tenth day. When complete healing has taken place, hot-air treatment is begun, which is continued each day for several months. Putti considers that stimulation of the muscles by electricity is more efficient than massage. Weightbearing is allowed in thirty days, aided by a simple mechanical apparatus. This is a critical period, and the joint may be stiff and a little painful. Care must be taken at this period not to push the hot-air treatment too far, and it may be best to suspend it for a few days and cautiously resume it. He considers that success is most likely in elbow operations; knee, jaw and hip operations following in the order given.

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72. Beckenheimer: Deutsch. med. Wchnschr., June 2, 1922, p. 729.
73. Putti: Lyon chir., March-April, 1922, p. 590.

Roeren,[74] in an article on mobilization of the ankylosed knee joint, describes his technic and emphasizes the importance of stimulating and developing the extensor muscles in every way before the operation, and of exerting traction after the operation. He fashions the femoral condyles like a roller and then makes concave the upper end of the tibia, implanting a flap of fat tissue extending between the ends and up beneath the mobilized patella. His results make him feel that the indications for the operation are even wider than Payr suggests in his latest papers. His contraindications are extensive injury to the extensor apparatus. He considers that the prognosis in cases in which the ankylosis is due to purulent infection must always be doubtful.

Volkmann's Ischemic Contraction.—Soubeyran[75] reports an excellent result, in a boy of 10 suffering from this crippling affection, by resection of 2 cm. of the radius and ulna. The bone was removed subperiosteally at different levels in the two bones, the sutured periosteal cuff retaining the position. The plaster fixation held the elbow flexed, the hand in supination, and the fingers fully extended. The fixation was retained for twenty-five days, and function then resumed. The case has been followed for eight years, and function remains complete, with eventual shortening of the arm of 1.5 cm.

Wavelike Incisions for Contractures.—Ritschl[76] has employed a wavelike incision in the skin to avoid secondary contractions of the scar in operations for the relief of joint contractures, such as tenotomy of the hamstrings and Dupuytren's contracture. The scar permits stretching as does a spiral spring, and crosses and recrosses the contracted deep structures at single points, thus minimizing the danger of adhesion between skin and tendon.

Tendon Transplantation for Musculospiral (Radial) Nerve Injury.—Billington[77] has always obtained greatly improved function in his cases of muscle transplantation for this lack of musculospiral innervation, and he feels reasonably sure, with an intelligent patient and a favorable condition of the tissues, of securing a functional result closely approaching that of a normal hand. He employs a technic slightly different from that of Jones, McMurray and Starr. The pronator radii teres is sutured to the extensor carpi radialis longior and brevior, the flexor carpi ulnaris to the extensor minimi digiti, and the extensor communis digitorum and the flexor carpi radialis to the three extensor tendons of the thumb. He believes the index finger functions without a suture to its special tendon, and that he secures more perfect thumb

74. Roeren: Arch. f. Ortho.- u. Unfall.-Chir. **20**:36, 1922.
75. Soubeyran: Paris méd., June 10, 1922, p. 490.
76. Ritschl: München. med. Wchnschr., 1922, No. 2, p. 397.
77. Billington: J. Bone & Joint Surg., July, 1922, p. 538.

function if the extensor longus pollicis is included. He is careful to cover his sutures with fascia. The application, immediately following the operation, of a specially molded, accurately fitting long cockup splint with thumb piece, which will positively maintain the proper attitude of the fingers and thumb, he considers essential in the after-treatment, as is also patient reeducation of the muscles and physiotherapy, for a period of two or three months.

Postoperative Care of Flexion Contraction of the Hip.—Stewart [78] has devised a simple and practical method of maintaining adequate hyperextension of the hip after Soutter's operation of fasciotomy, by means of an adjustable wooden or metal frame. He justly emphasizes the importance of after-care in these cases, which are likely to develop unfortunate complications due to nerve stretching or circulatory disturbances.

[ED. NOTE.—The editors have employed this frame with much satisfaction.]

Repair of Tendons in Fingers.—Bunnell,[79] in a carefully prepared article, discusses the repair of old tendon injuries of the fingers and hand, illustrating it by reports of difficult personal cases, with convincing photographic records. The points which he considers fundamental in the technic are the atraumatic handling of the tendons and the reconstruction of the gliding mechanism. In repairing an old injury of a flexor tendon of the finger, it is not practical to suture the tendon in the finger, because the sheath has been obliterated and there is not sufficient room to surround the suture line with a free fat graft thick enough to allow motion. When the suture line pulls over two joints, it is necessary to remove entirely the damaged tendon from the finger and to replace it with a free tendon graft. The suturing of the tendon can then be done at its insertion, and in the palm or forearm, where adhesions will do less harm. When the sheath has been obliterated, it is necessary to transplant not only the tendon, but also its gliding mechanism, either sheath or paratenon. The repair of both sublimis and profundus tendon is too difficult, and it is sufficient to replace the latter only and sacrifice the former. Function is necessary to keep a tendon graft in good condition. Grafts may be obtained from the sublimis tendon, the palmaris longus, the long extensors of the toes (a branched tendon), tubularized triceps, fascia lata, etc. The suturing of the tendon must be very strong in order to permit the all important postoperative exercises, and for this purpose, the author has found it very advantageous to employ a special clamp for holding the tendon

78. Stewart: J. Bone & Joint Surg., July, 1922, p. 548.
79. Bunnell: Surg., Gynec. & Obst., July, 1922.

and guiding the quilting sutures. It is difficult to remove a tendon neatly from a mass of adhesions, and especially from beneath the annular ligament and from within a finger, and for this purpose he has devised a special stripper. For placing a tendon with its paratenon through a tunnel in the finger, he uses a special type of flexible probe, with expanded middle and funnel-shaped rear end to carry the tendon. Quite often after a tendon repair or an infection or general traumatism, it is necessary to plane off the adhesions by the use of the tendon stripper, with immediate active movement following. Longitudinal incisions in the palm and the flexor aspects of the fingers should be avoided. Transverse incisions in the palm and lateral incisions in the fingers are better. Postoperative immobilization should not be employed. For a flexor tendon, the wrist may be strongly flexed and held by a check rein of adhesive plaster running up and around the forearm, robbing the muscles pulling against the tendon which has been operated on of their power to exert too much traction, but allowing active movement. This may be maintained for five weeks, until union is solid. Cooperation by the patient is essential, and he must be willing to undergo some punishment to obtain a good result. The prognosis should be guarded, but it is usually possible to restore from 80 to 95 per cent. of lost function.

[ED. NOTE.—All who have attempted the repair of old injuries of the tendons of the fingers have been impressed by the great difficulties in the way of obtaining good functional results. Bunnell has been working on this problem for five years. He has elaborated a plan of operation and a technic which are logical and have produced unusually good results. The specially devised instruments are ingenious and seem to serve a useful purpose. The article is difficult to abstract fully and we commend its perusal and study to all those called on to treat these difficult cases.]

PERIPHERAL NERVES

Late Affections of Ulnar Nerve Following Old Fractures.—Lusena [80] reviews the literature and reports two cases of ulnar paralysis developing eight and eighteen years, respectively, after a fracture of the humerus. Fractures of the capitellum resulting in a valgus position of the arm seem most likely to be followed by this affection of the nerve. This is probably because extra tension is brought on the nerve (perhaps by growth), or the groove is so altered that the nerve is exposed to injuries. Electricity, massage, the use of protective apparatus and operative transposition of the nerve have been tried with varying results.

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80. Lusena: Chir. de. Org. di Mov., April, 1922.

[Ed. Note.—One of the Editors has operated in three such cases, all following fracture of the external condyle. In all cases, the nerve has been transposed to the front of the joint. In two of the cases, the paralysis occurred in adult life following fractures in childhood; in the third, it came on between two and three years after the fracture. In one case, nearly complete restoration of function took place; in the second, great improvement followed the operation, and in the third no change has thus far resulted.]

Direct Stimulation of Peripheral Nerves.—Feiss,[81] from his experience with surgical repair of nerve injuries in several hundred cases at the American Ambulance, is convinced of the value of direct stimulation of the exposed nerve at the time of operation, in determining the procedure to be employed and the degree of injury to conduction. Many possibilities of technical error exist in the use of the method, and he formulates rules for their avoidance. 1. The nerve should be freed for a considerable distance to give free access, but care should be taken to avoid unnecessary exposure, in order not to injure the circulation of the nerve sheath. 2. The effects of mechanical stimulation during the freeing of the nerve should be carefully observed. 3. Before faradic stimulation, the nerve should be isolated by lifting it on a nonconductor hook, preferably of glass. 4. The nerve should be moistened constantly, but it should not lie in a saline bath, as this may cause spread of the current. 5. Bipolar electrodes are preferable to unipolar ones. The platinum points should be long enough to permit their relative positions to be altered by bending. 6. Such a current as would be just perceptible when applied to the tip of the tongue should be employed, beginning with a current of moderate strength. 7. Stimulation should be exerted on all sides of the nerve, but if there is a neuroma, the proximal segment is the important part in an efferent nerve, and the distal segment in an afferent nerve. 8. If a positive response is obtained, the nerve should be separated into its constituent bundles and tested individually. 9. The precise nature of the responses should be noted. 10. Different nerves respond in various ways to different frequency of current interruptions. Slow interruptions should be the rule in the beginning, increasing as necessary. 11. If it is decided to excise the neuroma, the motor response to the direct mechanical stimulation during the procedure should be observed. The entire distal portion of the limb should be sterilized, along with the operative field. This should be left undraped in order that the various phenomena following stimulation may be observed.

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81. Feiss: Surg., Gynec. & Obst., June, 1922, p. 243.

DISLOCATIONS

Retrosternal Dislocation of the Clavicle.—Schlegel [82] describes a very rare case of retrosternal dislocation of the clavicle. He states that only twenty cases have been reported. The right clavicle was displaced 4 cm. behind the sternum, causing symptoms of pressure on the innominate artery. Replacement was impossible, and a resection of the displaced end, 3 cm. in length, was performed, with an excellent functional result.

Anderson [83] reports a dislocation of the fourth cervical on the fifth, the fourth body overlapping the fifth by 2 cm. There were no symptoms of cord pressure. The case was seen twenty days after the accident, a fall backward on the head. Reduction was attempted by traction of 20 pounds, and manipulation under ether, but failed. A plaster cast and a head support were applied, and good function was assured one year after the accident. The roentgenogram at this time showed ankylosis and partial absorption of the fourth cervical.

Lateral Dislocation of the Lumbar Vertebrae.—Hartley [84] describes an unusual traumatic dislocation of the first lumbar vertebra to the left of the second, in a man of 38, caused by a heavy stone falling on his back. The patient was operated on by Sir Harold Stiles. By forced flexion, the spinous processes were separated and the articular processes cleared from one another. The dislocation was then reduced. As there had been ligamentous tearing, an arthrodesis of the articular processes was performed. Simple dislocations of the lumbar spine are very rare, because in forced flexion the cancellous bone of the bodies yields more readily than ligaments of the spinous processes and neural arches. The only other case reported of lateral dislocation of the lumbar spine without fracture was that of Charles Bill in 1824. Exposure of the neural arches and articular processes undoubtedly facilitates reduction, and it is suggested that open operation will prove of value in reduction of spinal dislocations of the more common types.

Constantini and Duboucher [85] report a luxation of the second lumbar vertebra to the right and forward on the third lumbar, with complete paraplegia. Reduction, which was not obtained by traction in the Sayre suspension method nor by manipulation under ether, was secured by open operation. The authors state that this was not difficult. They recommend this procedure, as does Hartley in the article reviewed above. No improvement in the paraplegia resulted.

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82. Schlegel: München. med. Wchnschr., 1922, No. 14, p. 511.
83. Anderson: Northwest Med., July, 1922.
84. Hartley: Edinburgh M. J., July, 1922.
85. Constantini and Duboucher: Rev. d'orthop., Jan., 1922, p. 27.

Traumatic Dislocation of the Hip in Children.—Doelle [86] has encountered two cases of traumatic dislocation in children. He finds only thirty-eight cases reported in the literature. Twenty were of the iliac type, six of the obturator, three sciatic, two suprapubic and six not specified. The mechanism of the injury, the diagnosis and treatment differed in no way from those in dislocations in adults.

Roello [87] also reports two cases in children of 6. These were ischiatic dislocations, one of which became spontaneously iliac after rest in bed for a few days on account of the child's condition. Roello considers that the ischiatic dislocation, from the nature of the ordinary injury, is probably commonly primary, and the iliac position secondary.

Dislocation of the Knee.—Meadows [88] easily reduced a complete forward dislocation of the tibia on the femur in a man of 40, under ether, by traction on the semiflexed leg and backward pressure on the tibia. The patient insisted on leaving the hospital on the second day, wearing an elastic bandage. He returned to work on the twentieth day.

The 1921 Report of the U. S. Department of Labor and Industry tabulates fourteen cases of dislocation of the knee, with an average temporary disability of 25.1 days, and an average partial disability of 83.5 days. For this year, the dislocations of the knee represented 16 per cent. of all dislocations.

Recurrent Dislocation of the Patella.—MacAusland and Sargent [89] present a well illustrated article on recurrent dislocation of the patella, on the basis of sixteen cases treated by various operative methods. In two cases, conservative methods were tried, but eventually operation was necessary. In two, the operation of capsulorrhaphy was performed, in one of which it was a failure. The other was not traced. Eight were treated by the operative procedure of Goldthwait, and four by a slight modification of the method, with only one recurrence, later relieved by a correction of a rotation deformity of the femur following a supracondylar osteotomy. The authors give a reference to Goldthwait's article, but do not refer to the method as his. As originally described, this method consists of splitting the infrapatellar tendon longitudinally, detaching the outer or viciously pulling half from the tubercle of the tibia, passing this detached half beneath the intact inner or benign half, and suturing it subperiosteally to the tibia on the inner side. MacAusland and Sargent's modification, which they prefer, consists in detaching a small portion of bone with the outer half of the

86. Doelle: Arch. f. klin. Chir. **118,** 1922.
87. Roello: Chir. de. Org. di Mov., April, 1922.
88. Meadows: Northwest Med., July, 1922.
89. MacAusland and Sargent.: Surg., Gynec. & Obst., July, 1922.

tendon, and inserting it in a bony pocket prepared on the inner side of the tibia.

[ED. NOTE.—This suggested modification seems sound to the Editors, as offering perhaps a firmer attachment. The important point remains that, by this method of tenoplasty, occasionally supplemented by a correction of a commonly coexisting rotary knock knee deformity, recurrence of this crippling dislocation has been entirely prevented.]

FRACTURES

Fracture of the Capitellum and Trochlea.—Lindem [90] describes a rather rare injury to the humerus at the elbow (seventeen cases in the literature) in which the articular face of the bone is split off by a fracture through the cancellous bone. The cause seems usually to be a fall on the outstretched hand or a blow on the dorsum of the upper third of the forearm, the force being transmitted through the radius and ulna. As a rule, the fragment is displaced anteriorly within the capsule, so that the articular surface faces forward. Lindem considers that excision of the displaced fragment is usually necessary. In two of Lindem's three cases, this was followed by a good functional result. The third patient refused operation.

Fracture of Lower End of Radius Associated with Fracture or Dislocation of the Lower End of Ulna.—Homans and Smith [91] report six cases of these rather frequently associated injuries. They consider that the initial difficulty in reduction is due to several factors: 1. The loosening of the radio-ulnar articulation, which destroys the fulcrum, by which the forced inversion of the hand might pry loose and draw down the displaced lower radial fragment. 2. The pull of the pronator quadratus, drawing together the lower 2 inches of the radius and ulna, offering a most effective resistance to any leverage made. 3. The fact that straight traction can be made only on the hand, and not directly on the radial lower fragment. It is possible also that interposed muscle (pronator quadratus or supinator longus) may in certain cases prevent reduction; but this has not been demonstrated in the operations in the authors' series. The ultimate and postoperative difficulty in holding the fractured radius is undoubtedly due to the instability of the lower fragment of the ulna; not to the possible fracture of the ulna at the level of the break in the radius, but the tearing off of the ulna at its articulation with the radius, resulting in a rupture of the strong interarticular fibrocartilage. They consider that open operation is nearly always necessary to replace the radial fragments and that, in

90. Lindem: Ann. Surg., July, 1922.

91. Homans and Smith: Boston M. & S. J., Sept. 14, 1922, p. 401.

instances in which the lower attachment between the radius and ulna is severed, secondary displacement and deformity are prone to occur.

Backfire Injuries.—Bizarro,[92] from an analysis of the records and roentgenograms of 190 cases of backfire injuries, concludes that (1) the commonest single lesion is a crack through the epiphyseal line of the radius; (2) the commonest double lesion is a fracture through the epiphyseal line plus a fracture of the ulnar styloid; (3) the carpal bone most commonly injured is the scaphoid; (4) violence is usually of the indirect type.

Traumatology of the Carpus.—Bizarro [93] has also studied 123 cases of injury of the carpal bones, finding that in 106, or 86 per cent., the scaphoid was fractured. This was the only lesion in sixty-eight, or 55 per cent., and it was a combined lesion in thirty-eight, or 30.9 per cent. There were two cases of fracture of the semilunar, six of the os magnum, and four of the unciform. Only twenty-seven of the fifty-five simple fractures of the scaphoid were seen within a week of the injury; but Bizarro believes that in such cases the wrist should be fixed in hyperextension, with the thumb included, for four weeks. He thinks fractures of the scaphoid rarely unite in spite of prolonged immobilization, but if seen early and properly treated, result in no disability. Old untreated fractures are of bad omen.

Direction Traction on the Pelvis.—In injuries of the pelvis, Block [94] has employed direct traction by means of wire passed through one or both ilia, with excellent results. In combination with direct skeletal traction on the femoral condyles, Block believes the method applicable for old luxations of the hip joint, extreme overlapping of the femur and limb lengthening operations.

Fractures of the Neck of the Femur.—Two German articles by Schmieden [95] and Bonn [96] advocating radical operative treatment of almost all intrascapular fractures of the neck of the femur are worthy of note in contrast to a review of the results of treatment by the Whitman abduction method by Powers.[97] These German surgeons advocate nailing or pegging when the capsule is not too badly separated and the central fragment is attached to it. They propose excision of the head in all other cases, placing the trochanter in the acetabulum, striving in older cases for an ankylosis, and in the younger, for motion. These surgeons have evidently had no experience with the abduction method

92. Bizarro: Ann. Surg., July, 1922.
93. Bizarro: Surg., Gynec. & Obst., May, 1922.
94. Block: Arch. f. klin. Chir., July 21, 1922, p. 410.
95. Schmieden: München. med. Wchnschr., 1922, No. 14, p. 526.
96. Bonn: Arch. f. klin. Chir. **120**:298, 1922.
97. Powers: Wisconsin M. J., Aug., 1922.

and have perhaps been influenced by such reports as that of the British Fracture Commission covering several hundred cases treated by Buck's extension, long lateral splints, wire mesh, etc. Only 23 per cent. of these cases were considered to have favorable results, and Walker of the New York Bellevue Service and Scudder of Boston consider good only 12 or 13 per cent. of results by these methods. When we compare the results of the Whitman abduction method reported by Campbell, 95 per cent. good results in seventy cases; by Anderson, 95 per cent. in 200 cases, and by Powers, 93 per cent. in sixteen cases, ten of which were obtained in patients over 60 years of age, it would seem to indicate that there is no need, in the vast majority of patients of any age, of resorting to open operation.

Necrosis of the Proximal Fragment in Fracture of the Neck of the Femur.—Axhausen [98] has had an opportunity to examine histologically a specimen of a fractured femoral neck in a boy of 16. The fracture had occurred ten months before his death from an intercurrent disease. Clinically and roentgenologically, bony union had taken place. The histologic examination revealed a complete necrosis of the central fragment, with a partial transformation into living bone proceeding from the distal fragment. The necrosis of the articular cartilage was leading to an arthritis. If such a change was taking place in a youth, with apparent firm union, it is easy to explain the changes which occur so often in older cases under pressure of weight bearing, such as secondary displacement of the head and absorption of the head and neck.

Muscle Interposition: A Cause of Delayed Union in Fractures of the Femur.—Kidner and Lakoff [99] treated a series of seventy-one cases of fracture of the femur between January, 1921, and April, 1922. Open operation, except for sepsis existing before the case came into their hands or for débridement in compound fractures, has been performed by them for only one reason; namely, because they believed that the interposition of muscular or fascial tissue made end-to-end apposition impossible and would be likely to delay or prevent union. They conclude that (1) if the fragments of a fractured femur are not brought into actual bony contact, as demonstrated by roentgenograms taken in two planes, under properly applied traction within three or four days, the case should be viewed with suspicion; (2) if specially planned methods are not immediately thereafter successful, manipulation under an anesthetic should be the next step, with an attempt to disengage the fragments from the muscle, success being appreciated by clean-cut

98. Axhausen: Arch. f. klin. Chir. **120**:325, 1922.

99. Kidner, F. C., and Lakoff, C. B.: Muscle in Interposition: Cause of Delayed Union in Fracture of Femur, J. A. M. A. **79**:200 (July 15) 1922.

rubbing of the bony fragments on one another; (3) if after such manipulation and the reapplication of traction, apposition is still lacking, as shown by roentgenograms, open operation should be undertaken; (4) at open operation, the muscle or fascial bands are cleared, and if end-to-end apposition of the interlocking type is possible, only external splinting is necessary. If there is a tendency toward displacement of the fragments, the authors have employed Gallie's beef-bone plates and screws, Magnuson's ivory screws, and, occasionally, metallic plates and bands. They believe the application of these forms of internal splints is more simple and sure than bone grafting.

Spiral Fracture of the Tibia and Fibula.—Caldwell's [100] experience with spiral fractures of the tibia and fibula leads him to believe that it is only possible to obtain complete anatomic replacement by operative measures. Manipulative reduction, and, in two cases, heavy continuous traction by means of ice tongs applied to the malleoli, totally failed of satisfactory results. The author favors banding with the Parham Martin band. Postoperative splinting is considered unnecessary. He reports ten such cases of fracture, two of which were treated conservatively, with poor results, and eight by operation, seven with good results. In the latter group, two patients developed serious postoperative complications; one, a severe infection, later necessitating a resection of 3 inches of the tibia, and the second a nonfatal pulmonary embolism and phlebitis of both legs.

[ED. NOTE.—The Editors are unable to agree with Caldwell that reposition in spiral fractures of the tibia and fibula can be obtained only by open reduction. Their own experience with the treatment of such cases by the early application of continuous skeletal traction (a pin passed above the os calcis) in conjunction with the Thomas splint, has been so favorable that, in an active fracture service in a large general hospital during a period of two years, open reduction was necessitated only once among all the simple fractures involving the shafts of the tibia and fibula of whatever type.]

Posterior Dislocation of the Foot with Posterior Marginal Fracture of Tibia.—Thomas [101] contests the commonly held view that posterior dislocation of the foot occurs as a result of Pott's fracture, with widening of the ankle mortise. That it is a frequent complication of Pott's fracture is not disputed, but its occurrence is only made possible by a posterior marginal fracture of the lower articular surface of the tibia, which may exist alone, but is frequently associated with other ankle fractures. A posterior oblique fracture of the lower end of the fibula is also a frequent accompaniment. The displacement tends to

100. Caldwell: Ann. Surg., June, 1922.
101. Thomas: Surg., Gynec. & Obst., July, 1922.

recur unless, following reduction, the foot is locked in strong dorsiflexion. Turner attributes the erroneous conception of the lesion to clinical observations preceding the use of the roentgenogram, when the feature of posterior displacement of the foot was recognized but the accompanying marginal fracture was overlooked.

[ED. NOTE.—The Editors are inclined to agree with Thomas in this matter, as they have yet to see such a dislocation without accompanying marginal fracture. Nevertheless, the old teaching remains unchanged, that in ankle fractures we must always be quick to recognize and correct posterior displacement of the foot.]

Injuries to the Astragalus.—Lotsch [102] brings out two rather interesting facts in an article on these rather rare traumatic lesions; namely, that compression fractures of the astragalus often take place as a result of falls of a considerable distance, striking on soft ground; whereas, if the surface is hard, compression fractures of the os calcis occur. Extreme plantar flexion may bring about a fracture of the posterior portion, and in such cases, a helpful sign is likely to be severe pain and tenderness along the tendon of the flexor longus hallucis.

Metatarsal Fractures.—Both Jacobson [103] and Vogel [104] call attention to the frequently unrecognized fractures of the metatarsal bones, which may occur from slight trauma or from no known direct trauma. One of the resulting difficulties is the disturbance of the transverse arch. Special foot supports are necessary.

Calcium and Phosphorus Metabolism in Patients with Fracture.—Tisdall and Harris [105] make a report of several years' work on the problem of the deposition of bone in fractures. Their conclusions are as follows: 1. The inorganic phosphate content of normal serum from birth to 20 years of age is remarkably constant. At 20, it drops sharply to a lower concentration and is again remarkably constant at this level throughout adult life. 2. The drop in the phosphate content of the serum at the age of 20 corresponds to the period of cessation of bone growth. 3. During the period of union of fractures in adults, the phosphate content of the serum is raised to a level approximately the same as that present in childhood; i. e., the period of active growth. 4. There is a close parallelism between the concentration of inorganic phosphate in the serum and the deposition of new bone, during both the period of growth and the period of repair of fractures. 5. In addition to the increase which occurs in the inorganic phosphate content of the serum

102. Lotsch: Klin. Wchnschr., Feb. 2, 1922, p. 318.

103. Jacobson: Zentralbl. f. Chir., 1922, No. 4, p. 116.

104. Vogel: Zentralbl. f. Chir., April 15, 1922, p. 505.

105. Tisdall, F. F., and Harris, R. I.: Calcium and Phosphorus Metabolism in Patients with Fractures, J. A. M. A. **79**:884 (Sept. 9) 1922.

during the union of fractures, there is a local metabolic process at the site of the fracture, which is probably even more important for the production of union than the increased phosphate content of the blood serum. In some of the animal experiments, this latter phenomenon was especially brought out. In one animal in which the apposition of the fragments was by chance reasonably accurate, there was union; while in the others, there was nonunion. Administration of calcium phosphate or yellow phosphorus or cod liver oil showed a rise in the phosphate content of the serum similar to that seen in the clinical cases of fracture examined. The administration of the various drugs did not appear to modify these curves to any appreciable degree. As in the clinical cases, the calcium curves showed only slight alterations.

Outline of the Treatment of Fractures.[106]—In the January, 1923, number of the ARCHIVES OF SURGERY is published an outline of the treatment of fractures, representing the conclusions of a group of general, orthopedic and industrial surgeons of large experience in the treatment of fractures in civil and military practice. The purpose of this conference, held in Boston under the auspices of the Fracture Service of the Massachusetts General Hospital, was to ascertain whether such a group, without previous consultation and trained in different schools of experience, could agree on such an outline of treatment. Somewhat to the surprise of all those present, it was found that, except for certain minor and generally unimportant details, they agreed. The outline above referred to presents their findings. This is not an attempt to standardize the treatment or to discourage in any way new methods or hold the experienced surgeon to any single method, but rather to place a helpful outline of treatment of proved worth in the hands of the general practitioner, who, as the industrial compensation cases show, is at present often giving his patients with fractures neither sound initial nor sound after treatment. For this purpose, as well as in the teaching of treatment of fractures, this well illustrated outline will prove, we believe, very useful.

AMPUTATIONS

Kineplastic Amputation of Forearm.—Bosch Arana[107] of Buenos Aires has written on the subject of kineplastic amputation of the forearm. He has been able to produce three active motors in the stump of the forearm, and hopes to be able to develop a prosthesis in which one motor will flex the fingers, the second extend them, and the third control the movements of the thumb.

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106. Outline of Treatment of Fractures, Arch. Surg. **6**:172 (Jan.) 1923.
107. Bosch Arana: Surg., Gynec. & Obst., Sept., 1922, p. 347.

[ED. NOTE.—That utilization of the muscles and tendons of an amputation stump to activate a prosthesis is possible had been demonstrated repeatedly. However, this work is still in an experimental stage, and much is yet to be done to make it practical. The chief difficulty up to the present has been the failure to develop appliances of sufficient mechanical perfection to be really useful. Considerable work is being done on this subject in Europe, and especially in Italy, where Putti has at his disposal admirable workshops and skilled mechanics. The Editors believe that the problem will be solved, but only when the surgeon is working in absolute harmony with skilled appliance makers who can carry out his ideas. Kineplastic operations performed under other conditions are at present bound to result unsatisfactorily, and this is likely to throw discredit on the entire procedure.]

INDUSTRIAL SURGERY

Acute Painful Back Among Industrial Employees Alleging Compensable Injury.—Conn,[108] after a rich experience with the problem of "backache" in industry, is convinced that the first duty of the surgeon is to make a correct diagnosis. After analyzing 156 cases, he suggests the following diagnostic classifications: 1. Traumatic strain. 2. Postural strain. 3. Sprain. 4. Sacrolumbar lesions. 5. Sacro-iliac lesions. 6. Infectious myositis. He believes trauma is not the important etiologic factor in a large percentage of backaches from alleged trauma. On the other hand, malingering, he believes, is uncommon, although implied to the surgeon by the patient's conception of the etiology and his overanxiety to obtain recognition of a real disability. Persons presenting osseous abnormalities in the lower portion of the spine are a potential group, especially susceptible to violence, but capable of developing disability unexcited by trauma. Conditions of sacro-iliac relaxation are of infrequent occurrence in industry. Sacrolumbar lesions include two classes as regards involvement of the articulation, extrinsic and intrinsic, the latter representing the grave traumatic lesions of most common occurrence.

[ED. NOTE.—The Editors believe that this analysis of Conn, based on experience and careful observation, is valuable. They find themselves in essential accord with his conclusions.]

Back and Foot Strain in Industry.—Wilson,[109] in a well illustrated article, reviews the etiology of these conditions from the standpoint of anatomy, physiology and body mechanics, outlining his ideas as to methods of diagnosis and treatment. He makes an important suggestion which, if adopted, would, in the opinion of the Editors, materially

108. Conn, H. R.: Acute Painful Back, J. A. M. A. **79**:1210 (Oct. 7) 1922.
109. Wilson: J. Indust. Hygiene, July, 1922.

diminish the number of chronic and discouraging cases of disability arising in industry. This suggestion is that, as a preventive measure, those who seek employment should undergo a routine physical examination by a well trained surgeon, who would grade them on the basis of body mechanics and susceptibility to strain. Those with marked physical defects should be required to undergo a period of corrective treatment with a view to improving their physical condition, permanent employment and higher wages being the reward of success. This would be applying to industry in a direct and entirely practical manner, the lessons learned in the war.

[ED. NOTE.—The Editors believe it would be a measure of economic importance also. Prevention is generally less expensive than cure, and an emphasis would be placed on physical fitness which would react favorably on the attitude of the employee toward his employer.]

Traumatic Osteitis of the Wrist.—Rogers [110] reports four cases of rarefying osteitis of one of the carpal bones, the semilunar in three and the scaphoid in one. The condition has previously been described by Preiser, in 1910, who reports five cases; and by Guie in 1914, who reports seven personal cases, and who collected twenty-nine cases from other clinics. Preiser believes the lesion to be a rarefying osteitis from interference with the nutrient artery, while Guie considers the condition essentially a compression fracture analogous to the so-called Kümmell's disease. In Rogers' cases, the onset of symptoms was gradual, generally covering a period of several months. There was definite pain referred to the wrist joint, brought on by use and relieved by rest. There was a slight amount of swelling associated with tenderness to pressure over the diseased bone. The limitation of motion occurred at first almost wholly in the direction of dorsiflexion. There was a question of trauma in all of the cases, but the symptoms began only after a considerable interval. In all, the roentgen ray showed the diseased bone "crinkled" in outline, smaller in size than normal, and with changes in the internal architecture. All of the cases were treated by excision; but two, only after a long period of conservative treatment. These two patients were relieved by conservative measures as long as they did not use the wrist, but the symptoms recurred as soon as they returned to their occupations. All the specimens were examined pathologically, and no evidence of tuberculosis was found. The cortex was thinned; the medulla contained much more fat tissue than normal, and there was loss of the normal bony trabeculae. The end-results are known in two of the cases and are reported as excellent. The writer suggests the name of traumatic osteitis of the wrist.

110. Rogers: Boston M. & S. J., June 1, 1922, p. 730.

RESEARCH

The Free Transplantation of Fascia and Tendon.—Gallie and LeMesurier [111] have conducted a long series of experiments to test the value of fresh autogenous transplants in taking the place of ligaments and tendons. These transplants must be carefully sutured and interwoven to insure freedom from slipping, and the successful methods they have devised are illustrated. They report the late results in fifty cases. In every case, the transplanted tissue has held, and has fulfilled the purpose for which it was transplanted. This they attribute to their care in bringing raw surfaces of the transplant in contact with firm connective tissue. Union has seemed complete in three weeks. No silk or unabsorbable tissue has been employed in their sutures. They quite properly observe that it seems more natural to use as ligaments material which we know will live and retain its normal characteristics.

[ED. NOTE.—This work of Gallie and LeMesurier seems to us so careful and thorough, both from the point of view of an experiment and from the end-results. Their methods should have wide adoption, with assurance that success will be achieved if the principles of their technic are followed.]

Experimental Production of Spasmodic Torticollis in Animals.—Rosenow [112] has obtained ticlike movements of the head and neck in animals as a common sequel to an encephalitis caused by the intercerebral infection of a somewhat atypical streptococcus isolated from cases of encephalitis. In these animals, the movements ceased during sleep, as the movements of a spasmodic torticollis do in human beings. They were also made worse by various forms of excitation. The type of movements in animals and in human patients were very similar.

Pathology of Osteitis Deformans: Paget's Disease.—Cone,[113] after extensive discussion of this subject, inclines to the belief that the changes in Paget's disease may be explained by a primary pathologic condition of the blood vessels like that observed in hereditary or acquired syphilis, with its accompanying bone changes, and such as is found on a small scale in chronic passive congestion or infections of a chronic character. He considers it not at all improbable that malaria, typhoid, influenza, erysipelas or parasitic organisms might induce such vascular lesions and be associated with a local or general osteitis deformans.

Fetal Chondrodystrophy in a Calf.—Seifried [114] examined all the endocrine glands in a calf showing all the characteristics of a fetal chondrodystrophy. All the glands were hypoplastic, the most marked

111. Gallie and LeMesurier: J. Bone & Joint Surg., July, 1922, p. 600.

112. Rosenow, E. C.: Experimental Production of Spasmodic Torticollis in Animals, J. A. M. A. **78**:752 (June 3) 1922.

113. Cone: J. Bone & Joint Surg., Oct., 1922, p. 751.

114. Seifried: München. med. Wchnschr., 1922, No. 11, p. 417.

changes occurring in the thymus, the cortex of which was markedly increased, with a diminished number of Hassall's bodies. The eosinophils and plasma cells were also much diminished, indicating also a hypoplasia of the thymus.

Observations on the Normally Developing Knee.—Cohn,[115] continuing his already reported studies of the ossification of the bones of the elbow and shoulder, now reports his observations on the growth of the bones of the knee. The epiphyses of the lower end of the femur and of the upper end of the tibia have attained the size of half the width of their respective diaphyses at 1 year. There is evidence of ossification within these epiphyses at a much earlier time. During the first four years, no other epiphyses are evident roentgenologically. At 5 years and 4 months, ossification has proceeded to such an extent in the lower end of the femur and the upper end of the tibia that it almost covers their respective diaphyseal ends. At this age, for the first time, there is ossification in the upper epiphysis of the fibula and within the patella. At 8 years of age, the depressions for the semilunar cartilage and the spines of the tibia are evident roentgenologically. By the tenth year, there is a close approximation between the respective epiphyses and their diaphyses. At 10 years and 11 months, we see the first evidence of the tubercle of the tibia. It is represented by a downward projection from the upper epiphysis of the tibia. During the twelfth year, the tubercle increases rapidly in size, and projects forward, being separated by a wide space from the shaft of the tibia. Union between the tubercle and the shaft proceeds from above down, and from before backward. At 18 years, this tubercle is completely ossified and united to the shaft. In one instance, the tubercle of the tibia seems to be ossifying through two separate centers. At 15 years and 4 months, there seems to be complete ossification within the epiphyses, and union of the respective epiphyses and their diaphyses. After the fifteenth year, there seems to be complete ossification of epiphyses and diaphyses.

Bone Atrophy.—Brooks [116] studied the process of bone atrophy in material obtained from patients suffering from poliomyelitis, traumatic nerve injuries, tuberculosis of bone, fracture, acute pyogenic osteomyelitis, deformities acquired and congenital and other conditions in which one extremity had not been used; also bones of animals in which function of one extremity had been impaired by nerve section, joint injury, or simple fixation by a plaster cast. In all instances, the bones of the nonused extremity were compared with the bones of the used extremity. The bones were studied in gross, by roentgen ray, microscopically, chemically and physically. In all instances, the changes were the same,

115. Cohn, Isadore: Normally Developing Knee, Arch. Surg. **5**:395 (Sept.) 1922.

116. Brooks: South. M. J., Oct., 1922, p. 823.

regardless of the cause of the nonuse, thus indicating that there is no necessity for assuming that any factor other than lack of function was responsible. In other words, the bone atrophy that follows a nerve injury is not the result of "trophic" influences, and that following an acute osteomyelitis is not the direct result of the toxins of the organisms; but in each instance, atrophy is the result of lack of function in the extremity, just as it follows the simple fixation of a normal extremity in a plaster cast. Bone changes make their appearance in a very few days after the cessation of function of an extremity. In the experimental animal, well marked changes are found after ten days of nonuse. The changes progress rapidly and are very marked in from ten to twelve weeks. Apparently, a maximum is reached in from six to twelve months, beyond which point there is very little change. In the adult, nonuse makes the bones become lighter and more fragile. The length is unchanged. The diameter is only slightly decreased. The diameter of the medullary canal is markedly increased. The trabeculae of the cancellous bone of the epiphyses become much thinner, and in many areas they disappear completely. After long periods of nonuse, the compact bone of the shaft becomes cancellous. The chemical composition of the entire bone is changed markedly. There is loss of mineral constituent and an increase in the proportions of organic material. It therefore follows that lack of function affects the size, shape, weight, roentgen-ray appearance and strength of bones by a change in the amount of matrix present, but the characteristics of bone tissue are not changed. The change is quantitative and qualitative. The end-result of constant lack of function during the growing period of a bone of an extremity is a bone which is relatively shorter and smaller. The relative size of shaft and epiphyses is changed, in that the shaft is relatively smaller than the epiphyses. The densities of the compact bone of the shaft and of the cancellous bone of the epiphyses are about normal. The chemical composition and physical characteristics of the bone are the same as in normal bone. The property of regeneration of bone is a property of the bone cell and not of the bone matrix. From a clinical study of the repair process in atrophied bones and a careful study of transplants of bones in animals in which transplants of atrophied bone have been compared to transplants of nonatrophied bone, it was not possible to detect any difference in the regeneration power of these two types of bone transplants.

Pathologic Changes in Muscle as a Result of Disturbance of Circulation: Experimental Study of Volkmann's Ischemic Paralysis.—Brooks [117] has also conducted a very complete and interesting series of

117. Brooks: Pathologic Changes in Muscle as a Result of Disturbances of Circulation: An Experimental Study of Volkmann's Ischemic Paralysis, Arch. Surg. **5**:188, July, 1922.

animal experiments to determine the effects of various types of circulatory obstruction on muscle, with special reference to the cause of Volkmann's ischemic paralysis. A review of the literature on this subject permitted the conclusion, based on clinical evidence, that there is a condition of paralysis of the muscles which appears, following circulatory obstruction, and which is independent of injury to the nerves. The clinical appearance suggests the presence of gross changes in the muscle, and pathologic examination of the tissue by other observers revealed the muscle cells in large part degenerated and replaced by fibrous tissue. The various experiments which were conducted and the results obtained were as follows:

EXPERIMENT 1 (Three animals).—The circulation of the leg was obstructed by application of a tight bandage above the elbow for periods varying from five to twenty-four hours. In one animal, no effect was produced on muscle function, and in the other two the limb was paralyzed, but the cause was found to be an injury to the nerves from direct pressure.

EXPERIMENT 2 (Three animals).—Permanent obstruction of the primary arteries of the limb was obtained by ligation of various arteries. In two animals, this resulted in complete temporary loss of muscle function, associated with superficial gangrene. Pathologic examination showed the muscle either pale and totally necrotic, or else of healthy appearance and with contractile response. The areas of muscle degeneration were sharply defined and the inflammatory reaction was slight. Between the extremes of normal muscle and totally necrotic muscle, there was no intermediate area in which the muscle still lived but could not function. One animal showed the phenomena of typical intermittent claudication, which disappeared at the end of three days, when a collateral circulation was reestablished.

EXPERIMENT 3 (Three animals).—Temporary obstruction of the primary arteries of the limb was obtained by ligation of certain arteries combined with the application of clamps to others, the latter being removed at the end of periods varying from six to twenty-four hours. Arterial obstruction of six hours resulted in temporary interference with function, followed by rapid recovery on removal of the clamps. Obstruction for periods of from seventeen and twenty-four hours resulted in complete paralysis below the knee, but the nerves were uninjured. Pathologic examination revealed sharply defined areas of degeneration in the muscle, accompanied by edema and hemorrhage; but the tissue was either completely necrotic or completely viable, and inflammatory reaction and fibrosis were lacking.

EXPERIMENT 4.—This was a series of experiments with isolated muscle. The same procedure of isolating the muscle (rectus femoris) was followed throughout. By operation, the muscle was completely freed from all surrounding tissue except at its origin and insertion, and these attachments were then ligated. The artery, vein and nerve were isolated from the surrounding tissue and preserved for treatment thus: A. Section of nerve (artery and vein intact). This resulted in paralysis of the muscle. At examination, the muscle fibers were found diminished in size and showing loss of definition of striation.

There were occasional degenerated fibers. There was no evidence at the end of a month of inflammatory reaction or fibrosis. The process was one of atrophy rather than degeneration. B. Injection of blood into muscle. In two animals, the artery, vein and nerve were left intact, and the muscle was distended by the injection of blood. In another animal, the artery was, in addition, ligated. No abnormality in the muscle reaction resulted, and at the end of one month there was no evidence of inflammatory reaction or fibrosis. C. Ligation of artery (vein and nerve intact). There were eleven experiments, the animals being killed at intervals ranging from seven to seventy days. The muscle showed normal physiologic properties in all instances, and in none did the muscle become fibrous. There were slight degenerative changes occasionally, but no inflammatory or fibrous reaction. In five other animals, the artery was ligated, and in addition the general circulation of the limb was diminished by ligation of various branches of the aorta. This resulted in edema of the muscle, but only slight degenerative change and inflammatory reaction. Further diminution of the blood supply of the limb resulted in complete absorption and disappearance of the muscle. D. Ligation of vein alone. There were nineteen experiments, the animals being killed at periods varying from one to 120 days. The procedure resulted in a constant pathologic change in the muscle. This change was characterized by an initial period of acute inflammatory changes in the muscle and subsequently by the development in the muscle of an extensive fibrosis. The fibrosis in some instances replaced a large portion of the muscle fibers and decreased very markedly the power of the muscle. In other instances, the substitution of the muscle fibers by fibrous tissue was complete.

The constancy of the pathologic process and the certainty with which it follows the experimental method described are important points. The author concludes from this large series of experiments that a muscular weakness associated with a certain stiffness which follows the occlusion of a large artery may be a phenomenon of ischemia. It would be likely to improve with the development of a more adequate circulation. A complete paralysis following the occlusion of an artery is evidence of an anemia, and usually signifies impending gangrene. Anemia and inflammation are incompatible processes. Any condition which develops within from twenty-four to forty-eight hours and which is characterized by heat, swelling, pain and a subsequent fibrosis cannot be the result of permanent occlusion of an artery. That the classic picture of Volkmann's ischemic paralysis could be explained only on the basis of acute venous obstruction would seem quite clear. Probably, some of these cases may be the result of pure local pressure necrosis analogous to the decubitus ulcer or pressure sore over the heel from continuous pressure, but it should be a simple matter to differentiate such cases from the others.

MISCELLANEOUS

"Flat Hand."—Goldthwait [118] again calls attention to the importance of the carpal arch, describing carefully the anatomy of this structure,

118. Goldthwait: J. Bone & Joint Surg., July, 1922, p. 469.

and the necessary part which it plays in proper functioning of the hand. When this arch "flattens," the palmar tendons are no longer held in place by the hook of the unciform and the trapezium, and weak and painful action results. By a light wrist strap with two pelots, one on either side of the trapezium, and a thumb loop to retain the strap in place, the arch may be restored and function rendered painless.

Tennis Elbow.—Romer [119] maintains that there are three types of the so-called tennis elbow: 1. The commonest type, in which a condition analogous to a tenosynovitis exists in the muscles attached to the condyles, usually the external. He considers that this is caused by the frequent jerks of the muscles at their origin, and he speaks of it as a traumatic fibrosis. 2. A type in which the tender point is on the condyle itself, owing to irritation of the periosteum, suggestive of a minute tear of some muscle insertions. 3. A synovitis of the radio-ulnar joint. These three varieties, Romer suggests, may be stages rather than actual types. Romer believes that the use of a large handled or heavy raquet is the commonest cause, unless the player has a correspondingly large hand or strong arm. His treatment consists of massage for from ten to twelve days and strapping of the forearm in such a way that the pull of the muscles on their origins at the external condyle is checked. Play should be begun with this strapping in place.

[Ed. Note.—We recognize that there is a condition more or less accurately described as epicondylitis, in which the tenderness is over the bony condyle, and the roentgen ray may show evidence of periosteal reaction and lime salt deposit. This condition is relatively rare, and is not induced, we believe, by tennis or other sports or occupations in which muscle action and not direct trauma is the etiologic factor. We believe that the hypothesis of a traumatic fibrositis, analogous to a tenosynovitis, at the origin of the muscles attached to the external condyle, is not necessary; nor is more than clinical evidence advanced by Romer to bear it out. Neither have we ever found any evidence of a true radiohumeral synovitis. When the muscles originating from the external condyle and the supinator brevis are relaxed the motion of this joint has in our cases been painless. The condition seems to us more satisfactorily explained by a bursitis in the small bursa, demonstrated by Osgood [120] to be usually present beneath the conjoined tendon and supinator brevis and directly over the radiohumeral joint. This bursa has been found distended in cases of this affection at operation, and its evacuation and curetting have been followed by relief of symptoms.

119. Romer: Lancet, July 8, 1922.

120. Osgood, R. B.: Radiohumeral Bursitis, Epicondylitis, Epicondylalgia (Tennis Elbow): A Personal Experience, Arch. Surg. **4**:420 (March) 1922.

Myositis Ossificans.—Ferrarini [121] reports nine cases of myositis ossificans, five of them in the region of the elbow, three of them following dislocations. The ages varied from 15 to 30 years. The tumor mass grew smaller under rest in all cases, but in none did it disappear. He believes the condition is due to a chronic irritation of the interstitial tissue.

Isolated Disease of the Head of the Second Metatarsal Bone.—Axhausen,[122] in examining two specimens of that peculiar isolated disease of the head of the second metatarsal bone described by Köhler and others, found that the largest part of the head had become necrotic. In some portions, a fibrous substitution of the necrotic bone was seen, with an active reaction of the synovial membrane like that in the proliferative type of chronic arthritis. A direct traumatic etiology is suggested neither by the histories nor the histologic findings, and Axhausen is convinced that the process is analogous to osteochondritis dissecans, which the author believes to be caused by an embolic mycotic process.

Vaccine Therapy in Bone Lesions Due to the Typhoid Bacillus.—Vincent [123] has compiled thirty cases of typhoid and paratyphoid osteopathies treated by vaccine therapy, and adds one of his own as a convincing example of the efficacy of this form of treatment in many cases. His patient was a young woman who developed a series of abscesses, several osteomyelitic bone lesions and a mucomembranous enteritis during a convalescence from a fever supposed to be typhoid, but whose blood agglutinated the paratyphoid bacillus. She was given four injections, representing a total of 2,500 million bacilli, and all bone and other lesions promptly cleared up, although they had existed for three months before the vaccine therapy was instituted.

Lordotic Albuminuria with Pott's Disease.—Neukirch and Rottmann [124] describe an interesting case of a young woman with a large tuberculous kyphos, including the sixth to the eighth dorsal vertebrae, and a very marked lumbar lordosis in the upright position. When the patient was upright, there was albuminuria. This ceased promptly when the lordosis was corrected by posture. Ureteral catheterization proved beyond doubt that only the left kidney was responsible for the albuminuria, seeming to prove that the phenomenon has been correctly explained as due to a congestion of the veins in the left kidney.

Progress in the Care and Education of Crippled Children in Ohio Under New Laws.—Chollett's [125] report of progress in the development

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121. Ferrarini: Chir. d. org. di movimento, July, 1922.

122. Axhausen: Beitr. z. klin. Chir. **126**:451, 1922.

123. Vincent: Bull. de l'Acad. de méd., Paris, May 9, 1922, p. 517.

124. Neukirch and Rottmann: Klin. Wchnschr., March 11, 1922, p. 523.

125. Chollett, B. G.: Care and Education of Crippled Children, J. A. M. A. **79**:1297 (Oct. 14) 1922.

and organization of a movement originated and still fostered by the Rotary Clubs of Ohio for the care and education of the crippled children of the state is most encouraging. State laws have been passed; a large central committee has been formed to have supervision of the work, and educational propaganda has been started in order to bring to the attention of the public the advantages of proper treatment for various types of cripples.

Scope of Occupational Therapy and Training of Occupational Aides.—Brackett,[126] in discussing the scope of occupational therapy, describes the minimum requirements in the Boston School of Occupational Therapy for training in this rather new branch of medical work: (1) an age of from 24 to 40, with at least a high school or equivalent education, and (2) an occupational therapy school course of one year of twelve working months. Nine of these months are spent in the school, where applicants are taught major and minor crafts for practical work and are given courses of a varying number of lectures in anatomy, neurology, mental hygiene and diseases, orthopedic surgery, contagious disease, tuberculosis, syphilis, psychology, social service, physiotherapy, hospital ethics and management and personal hygiene. Three months of the twelve are spent in practical work in hospitals under supervision. After a year or more work in hospitals or with groups of patients, these graduates may apply for registration by the Massachusetts Society of Occupational Therapy.

Quackery, Miracle Healing and Medical Cults.—Lovett,[127] in an interesting article, reviews the history of quackery and miracle healing, and shows that irregular practice has always been and probably always will be with us in varying forms. In general, it follows well defined channels, as it rests on an inherent tendency in the human race to believe in the wonderful, the mystical, the obscure and the new. Today, Christian science and manipulative methods hold the center of the stage. If they were wiped out, some other form of irregular practice would take their place. Christian science developed under a wonderful woman who had a very practical understanding of advertising and business matters; but she is dead and her successors are quarreling. Osteopathy is in reality bone setting, with a pseudoscientific explanation superimposed; but chiropractic seems merely a cheap commercial enterprise modelled on osteopathy, too successful, too fast growing and too confiident to endure permanently, although at present it is a real menace to the health of the community. Miracle healing and mental healing, when associated in any way with religious belief, cannot be argued about,

126. Brackett: Arch. Occupational Therapy, June, 1922.
127. Lovett: Boston M. & S. J., July 15, 1922, p. 53.

and may only be restricted to the extent of preserving the health of the community and safeguarding the avoidable deaths. With regard to manipulative methods, we should adopt the course already adopted by *The Journal of the American Medical Association,* and instead of excluding the literature of irregular cults from our libraries, we should give it the widest publicity, and encourage students and practitioners to familiarize themselves with it. Secondly, we should teach our students the principles of physical therapeutics and show them why manipulative methods are at times beneficial. In order to do this, we should investigate these manipulative methods ourselves.

BOOK REVIEWS

ORTHOPAEDIC SURGERY. By Sir Robert Jones and Robert W. Lovett. New York: William Wood & Co., 1922.

This is a text and reference book of 700 pages, well illustrated. Under the heading of orthopedic surgery, there are included: joints and their affections; bones and their affections, including ununited and malunited fractures; disturbances of the neuromuscular mechanism; congenital deformities; static and other acquired deformities, and the principles and details of apparatus. We believe this is the most important work dealing with these subjects which has yet been written in any language. Two eminent clinicians and investigators of wide experience, the one British and the other American, have succeeded in writing a book which is judicial in its attitude, meticulous in the credit it gives to other men's work, conservative in its tone, but practical and authoritative in its statements. The book is a contribution to general as well as orthopedic surgery, the latter of which the authors recognize as only a special branch of the former. To orthopedic surgeons, and especially to teachers of orthopedic surgery, the book is most welcome.

BODY MECHANICS AND HEALTH. By Leah G. Thomas and Joel E. Goldthwait. Boston: Houghton, Mifflin Company.

The demand has long been felt for a book enunciating the principles which underlie the close association between proper use of the body and good health, and describing in detail the exercises and rest positions best calculated to bring about correct posture. This little book, well illustrated, and written quite as much for teachers of physical training as for physicians, is likely to supply this demand. It is a distinct contribution to orthopedic surgery, especially if one derives the word from παιδεύω and ὀρθός, "to educate straight."

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CONTENTS

PAGE

Cystic Disease of the Bones: A Study of Fifteen Cases. Astley P. C. Ashhurst, M.D.; Ralph S. Bromer, M.D., and Courtland Y. White, M.D., Philadelphia 661

Artificial Nerve Branches for Innervation of Paralyzed Muscles. Byron Stookey, M.D., New York 731

Preservation of the Facial Nerve in the Radical Treatment of Parotid Tumors. Alfred W. Adson, M.D., and William O. Ott, M.D., Rochester, Minn 739

The Treatment of Brain Abscess by the Induction of Protective Adhesions Between the Brain Cortex and the Dura Before the Establishment of Drainage. Charles E. Dowman, M.D., Atlanta, Ga 747

Primary Lymphosarcoma of the Prostate. Douglas Symmers, M.D., New York 755

The Physiology of Pulmonary Embolism as Disclosed by Quantitative Occlusion of the Pulmonary Artery. Gilbert E. Haggart, M.D., and Arthur M. Walker, Boston 764

Studies on the Calcification of Callus. Edwin P. Lehman, M.D., St. Louis 784

A Study of Diverticulum Formation in the Appendix. A. P. Stout, M.D., New York 793

Primary Carcinoma of the Male Urethra. Herman L. Kretschmer, M.D., Chicago 830

The Pathogenesis of Peptic Ulcer. J. E. Sweet, M.D.; L. T. Buckman, M.D.; A. Thomas, M.D., and E. M. Bell, M.D., Philadelphia 837

Calcification and Ossification of the Meninges. Albert E. Halstead, M.D., and Frederick Christopher, M.D., Chicago 847

Twentieth Report of Progress in Orthopedic Surgery. Robert B. Osgood, M.D.; Robert Soutter, M.D.; Harry C. Low, M.D.; Murray S. Danforth, M.D.; Herman C. Bucholz, M.D.; Lloyd T. Brown, M.D.; M. N. Smith-Petersen, M.D., and Philip D. Wilson, M.D., Boston 858

Index 909